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BENHA UNIVERSITY
FACULTY OF VET . MED .
DEPARTMENT OF ANIMAL MEDICINE

EQUINE MEDICINE

(SUMMARIZED INTEGRATED COURSE FOR 5 th YEAR STUDENTS)



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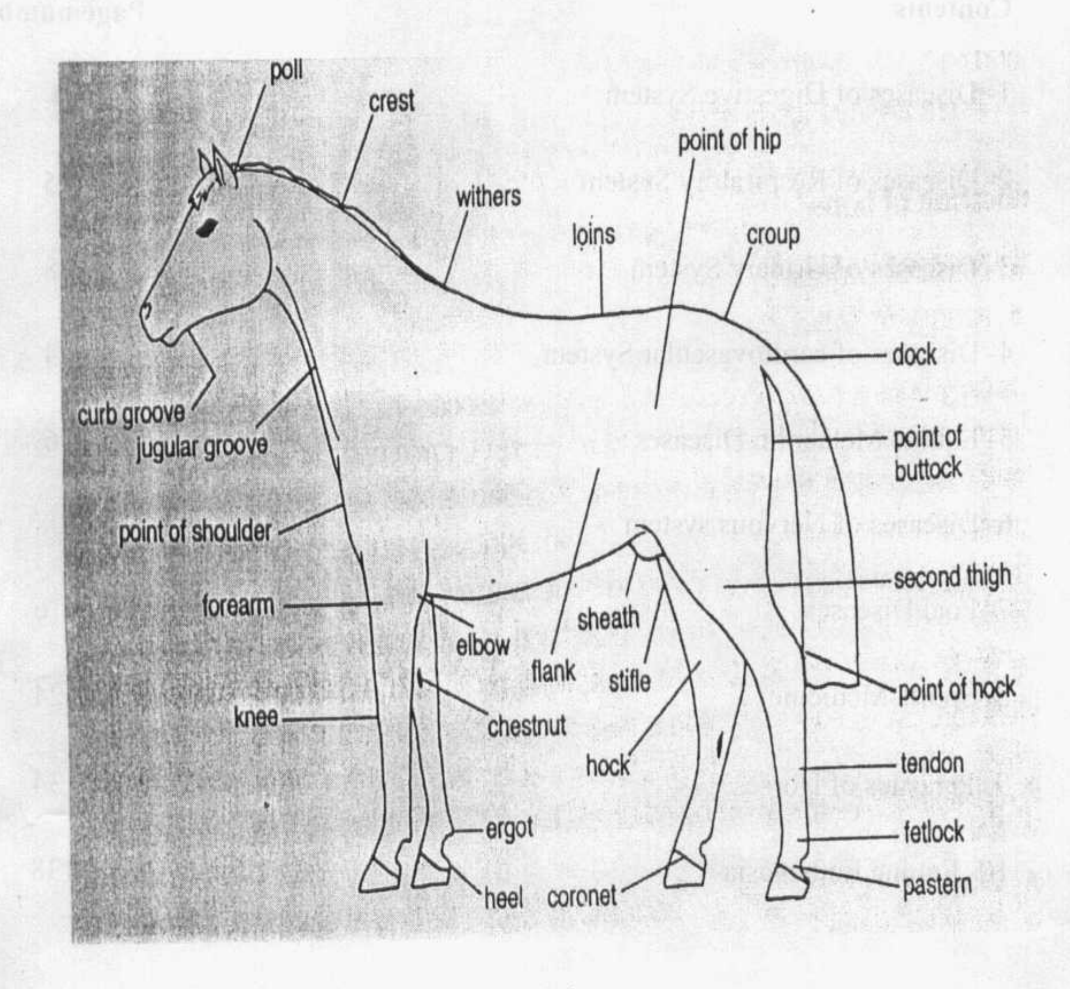




Table of Contents

Contents	Page number

DISEASES OF DIGESTIVE SYSTEM

AFFECTIONS OF ORAL CAVITY IN HORSES

(I)STOMATITIS IN HORSES

Definition :

Stomatitis means inflammation of oral mucosa and it include glossitis (inflammation of tongue), gingivitis (inflammation of gum) and palatitis (lampas). Clinically it is characterized by partial or complete loss of appetite, smacking of the lips and salivation.

Etiology :

Physical, chemical and infectious agents.

(A) Physical Agents:

1- Drinking of hot water.

2- Eating sharp awns or spines.

3- Foreign body injury.

4- Trauma while dosing.

5-Malocclusion of teeth.

(B) Chemical agents:

1- Irritant drugs administered in over strong concentrations e.g. chloral hydrate.

2- Licking of counter irritants e.g. mercury and cantharides compounds.

3- Irritant substances administered by mistake e.g. acids, alkalis, phenolic compounds.

4- Systemic poisoning e.g. chronic mercury.

5- Included in syndrome of uremia in horses.

(C) Infectious agents:

1- Vesicular lesions in vesicular stormatitis and herpes virus infections.

2- Lingual abscess caused by Actinobacillus spp.

Clinical findings :

1- Partial or complete anorexia.

2- Salivation and smacking of lips.

3- Slow, painful mastication.

4- fetid odour on breathing (if bacteria invade the lesion).

5- Enlargement of local lymph nodes (if bacteria invade the lesion).

6- Increased desire for water.

7- Toxemia may be present if stomatitis secondary to systemic disease.

8- Vesicular lesions are usually thin-walled vesicles 1-2cm in diameter filled with clear serous fluid. When vesicles ruptured, they leave sharp-edged shallow ulcers.

Treatment:

1- Isolation of affected animals and should and watered from separate utensils particularly if an infectious agent is suspected.

2- Frequent application of a mild antiseptic e.g. 1% suspension of sulfonamide in glycerin or 2% suspension of borax or 2% solution of copper sulfate.

3- inopleat ulcers require curettage or cauterization with silver nitrate stick or tincture of iodine.

4- Soft, appetizing food should be offered and feeding by stomach tube or I/V injections in severe prolonged cases.

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(II) LAMPAS (PALATITIS) IN HORSES

Definition:

Lampas means swelling and hardening of hard palate m.m. in horses (just behind upper incisor arcade) .

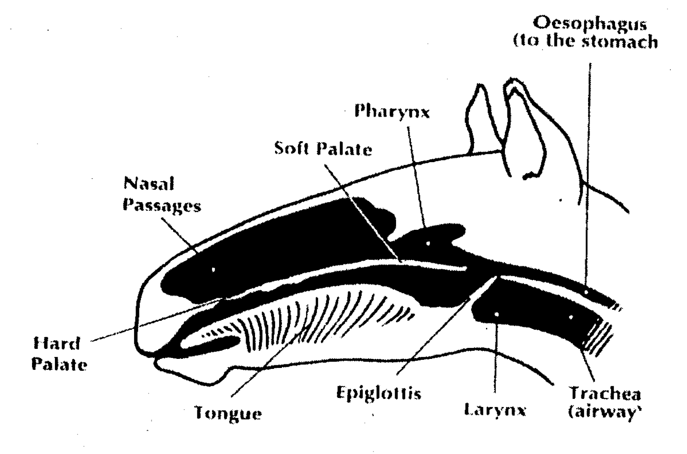


Fig. 5.4 Swallowing breaks the seal between the soft pal ate and the larynx.

Etiology :

(a) In young horses (Foals) :

Associated dentition i.e. change from temporary to permenant teeth.

(b) In mature horses :

Associated congestion of oral region .

Clinical signs:

1- Pain (during suckling and mastication).

2- Mouth inspection shows that anterior rugae of hard palate extend below the level of incisor teeth.

Treatment :

(a) In foals :

Recovery usually spontaneous and uneventful.

(b) In olders :

Condition persists for extended period, therefore feeding soft feed-laxative diet e.g.bran mash is recommended until recovery.

* * *

(III)CLEFT PALATE PALATOSCHISIS) IN HORSES

Definition:

Common congenital defect in newborn foals.

Etiology:

(a) Old evidence :

Hereditary cause.

(b)Recent evidence :

Viral infection or ingestion of toxic agents for pregnant mares during gestation.

Clinical signs:

1- Milk dripped from nostrils (During suckling).

2-Dysphagia.

3- Mouth inspection reveals the cleft palate.

N.B: Aspiration pneumonia due to inhalation of milk commonly occur.

Treatment :

Surgical correction (usually give a poor results).

* * *

AFFECTIONS OF PHARYNX IN HORSES

(I)PHARYNGITIS IN HORSES

Definition:

Inflammation of pharyngeal mucosa.

Etiology:

(a) Trauma (Common cause in horses) :

Foreign bodies e.g. : Awns , spines, wires ... etc.

(b) Infection (Bacterial and viral) :

- Bacterial infection e.g. Strangles, anthrax...etc.
- Viral infection e.g. Influenza, parainfluenza, rhino virus, viral arteritis, herps virus, aseno virus... etc.

Clinical findings:

1- Horse stand with head extended .
2- Refuse to eat or drink.
3- Painful swallowing (If forcely eating) .
4- Regurgitation of fluid and food through nostrils (In severe cases) .
5- Paroxysmal cough (positive cough test)
6- Enlarged retropharyngeal lymph node (Inflammed or abscessed) .

N.B.:

"Chronic Folliculr Pharyngitis Syndrom" commonly occurs in horses after viral infection accompnied by hyperplsia of lymphoid tissue in pharymgeal mucosa giving it granular-nodular appearance hanging from phryngeal roof .

Treatment:

1- Remove primary cause.
2- Parenteral antimicrobial (antibiotics or sulfonamides) .
3- Electrocautary (may be useful) .

* * *

(II) PHARYNGEAL OBSTRACTION

Definition:

Mechanical partial occlusion of pharyngeal cavity.

Etiology :

(a) Foreign bodies:

Corn cobs, bones, piece of wire ... etc.

(b) Tissue swellings:

1- Chronic follicular pharyngitis syndrome.
2- Retrophryngeal lymphadenitis or abscess (caused by strngles).
3-Goitrous thyroid.

Clinical findings:

1-Stertorous respiration.

2- Coughing.

3- Difficult swallowing.

Treatment:

1- Remove primary cuase.

2- Parenteral treatment with pencillin-streptomycin (Pharyngeal abscess) .

3- Surgical interference (Forieng bodies, pharyngeal cysts, goitrous thyroid).

* * *

(III)PHARYNGEAL PARALYSIS IN HORSES

Definition:

- Acquired absence of pharyngeal neuromuscular tone.

Etiology :

(a) Peripheral nervous system injury :

Glossophryngeal nerve injury due to :

1- Trauma (Throat region) .

2- Infection (Guttural pouch) .

(b) Central nervous system injury :

Secondary to specific CNS disease due to:

1- Rabies (encephalitis).

2- Equine pests (African horse sickness)

3- 3-Botulism (Cl.Botulinium).

Clinical findings:

1- Constant salivation.

2- Dropping of food from mouth.

3- Regurgitation food through nostrils.

4- Inability to swallow (with an absence of signs of pain) .

5- Coughing .

6- Dehydration.

7- Rapid loss of condition .

N.B.: Aspiration pneumonia is a common complications if left without treatment.

* * *

AFFECTIONS OF ESOPHAGUS IN HORSES

(I)ESOPHAGITIS IN HORSES

Definition :

Inflammation of esophageal m.m.

Etiology :

1- Trauma from foreign bodies (most common)

2- Injury caused by stomach tube.

3- Irritating chemicals (e.g : Breaking of capsule containing irritant drug).

Clinical findings :

In severe cases there are:

1- Extension of head and neck.

2- Salivation.

3- Dysphagia .

4- Regurgitation (via mouth and nostrils) .

5- Palpation reveal esophageal spasm (cervical musculture).

Treatment:

1- Withholding feed (relieves condition) .

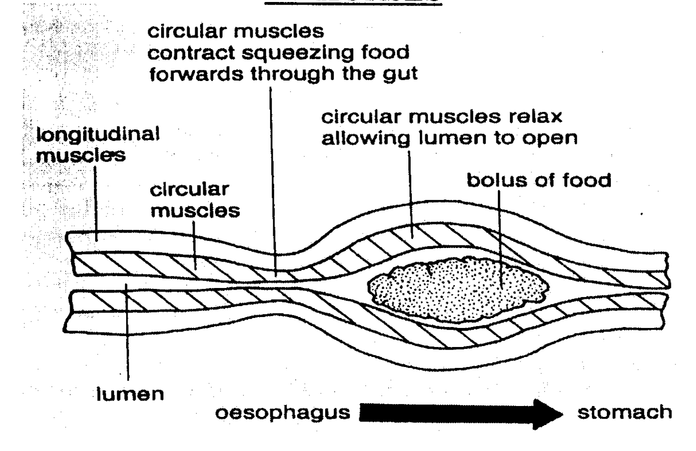
2- Antibiotics (control infection) .

3- Corticosteroids (Control pains).

4- Analgesics (control spasm) .

* * *

(II) ESOPHAGEUL OBSTRUCTION IN HORSES



Obstruction of esophagus my be acute or chronic, internal or external.

Etiology:

(A)Internal obstruction :

- Caused by swallowed materials like food mases or forgien bodies e.g :
  1- When horses allowed for eating dry hay (Or wood shavings bedding) . The bolus lodges at cardia causing obstruction.
  2- When horses accidentally swallowed nasogastric tube.

(B)External obstruction :

- Caused by pressure on esophagus by surrounding organs or tissues e.g.:
  1- Stomach carcinoma .
  2- Persistent aortic arch.
  3- Mediastinal neoplstic L.N..
  4- Mediastinal or cervical abscess.

Clinical findings:

(A) Acute obstruction (Choke) :

(a) In terminal part of thoracic esophagus (Common):

Obstruction can not be seen or even palpated .

(b) In thoracic inlet obstruction (Less common) :

- There is " **esophageal spasm and foreceful painful peristalsis and swallowing movements**" at site of obstruction .
- Generally, signs proceed as follow:
  1- Sudden stop eating.
  2- Anxiety and restlessness (Paw at ground).
  3- Forceful attempts to swallow (accompanied with coughing).
  4- Regurgitation of food through nastrils .
  5- Salivation.
  6- Impossible passage of nasogastric tube.

N.B.: Death may occur from subsequent aspiration , or when obstruction persists, from dehydration .

(B) Chronic obstruction:

There is an absence of acute signs:

1- The swallowing movements are usually normal until the bolus reaches.
   The obstruction when they re replaced by a more forceful movement.
2- The swallowed material either pass slowly through the stenotic area or accumulates and is then regurgitated.
3- In the gater stages , there may be no attempt to eat slid food but fluids may be taken and swallowed satisfactorily.

Treatment:

Treatment of chronic obstruction is usually unsuccessful . In acute obstruction the following may be useful .

1- Sedation of the animal first by an ataractic drug (chloropromazine Hcl 50-100 mg/kg) or chloral hydrate to help in relaxing the esophageal spasm (S/C injection of atropine sulphate 16-32 mg may be u seful)

2- Gentle attempt by passage of stomach tube to push theopush the obstruction onward (but avoid damage of mucosa which is probably fatal lesion in horses).

3- Surgical in terference (Eosophegotomy) but not highly successful.

N.B:

Accumulation of ingested material in lower esophagus of horse is more difficult to remove .

- Small quantities of warm saline should be introduced through a stomch tube passed to the point of obstruction and then pumped or siphoned out.
- This may be repeated a number of times until fluid comes clear .

* * *

III-ESOPHAGISM (SPASM OF ESOPHAGUS)
(ESOPHAGISM MOST COMMONTY IN YOUNG HORSES)

Etiology:

- The exact etiology is unknown, although the condition has been observed in the following cases:

1- Nursing foals when they begin to take solid food .

2- During routine stomch tube passage.

3- In horses with acute esophgitis and esoph. Obstruction .

Clinical findings:

Resemble those esophgeal obstruction

Treatment:

1- Atropine sulphate (0.03,0.07mg/kg B.W) to control the spasm, but morphin sulphte (60-90mg S/C) may be used to relieve severe spas.

2- Tranquillzers (Xylazine 1.1 mg/kg B.W.TV) to control nervous horses.

* * *

IV-ESOPHAGEAL DIVERTICULUM (DILATATION)

This condition assumes its great importance in horses

Etiology:

esophageal diverticulum occurs most often secondarily to esophageal stenosis i.e.associated indirectly to esophgeal stenosis i.e. associated indirectly with chronic obstruction and esophagitis.

Clinical findings:

Similar to those of esophgeal obstruction.

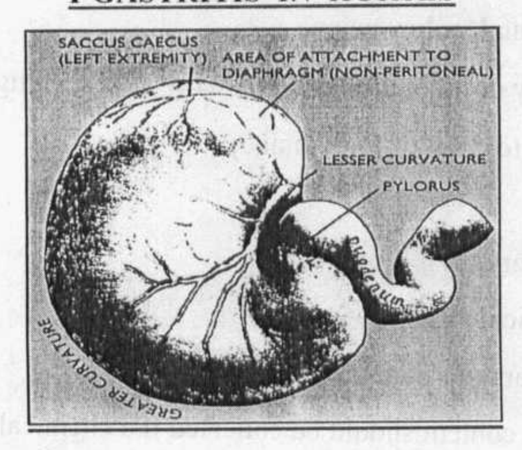
Treatment:

Surgical exposure of the diverticulum nd careful apposition of the esophageal musculture is the only effective treatment.

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DISEASES OF STOMACH IN HORSES

I-GASTRITIS IN HORSES



Definition:

Inflammation of stomach m.m

Etiology :

Physical, chemical or metazoan agents :

(A) Physical agents :

Ingestion of foreign materials e.g: sand (Wind-sucking horse).

(B) Chemical agents :

Accidental ingestion or licking of irritant caustic and toxic agents in e.g: arsenic, mercury, lead, copper .. etc.

(C) Metazoan agents :

Massive infestation with:

1- Gastrophilus sps. (Bot fly larvae) .

2-Habronema sps. (Muscae, Microstoma and Megastoma)

Clinical findings :

(a) Acute gastritis:

1- Reduced appetite (but excessive thirst).

2- Acute abdominal colicy pains.

3- Regurgitation (Severe cases) .

(B) Chronic gastritis :

1- Depressed or depraved appetite.

2- Subacute abdominal colicy pains .

3- Regugitation may occur sporadically (usually after feeding) .

4- Emaciation (due to lack of food intake).

Diagnosis :

(A) History

(B) Clinical signs

(C) Laboratory diagnosis

Sample of stomach content should be collected if a chemical poison is suspected.

Treatment:

1- Empty alimentary tract using paraffin mineral oil (to avoid further irritation to mucosa if saline purgative is used).

2- Gastric lavage with isotonic saline every 4 hours (if chemical poisoning is suspected) .

3- Gastric sedative e.g: Buscopan composotum.

4- Anti-acids e.g: Magnesium hydroxide (or carbonate).

5- Soothing therapy e.g: kaolin or pectin (Sometimes with charcoal) .

N.B: During convalescence, soft palatable, highly nutritional food e.g. green feed and bran mashes are recommended.

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II-GASTRIC ULCERATION IN HORSES

Definition:

Means ulceration of stomach m.m.

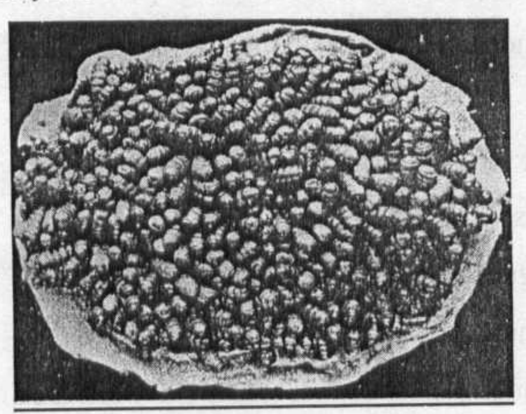
Etiology :

1- Parasitic gastritis (Gastrophilus and Habronema megastoma).

2- Repeat dosing with non-steroidal anti- inflammatory drugs **(NSAIDs)** .

3- Diets high in concentrates play a role.

N.B: In foals disese may associated viral enteritis caused by rotavirus infection



Pathogenesis :

- Gastric ulceration commonly results in :

1- Pyorous spasm. 2- Increased gastric motility.

3- Gastric hemorrhage. 4-Perforation and peritonitis.

N.B: Healed ulcer may cause pyloric or duodenal partial or complete obstruction.

Clinical findings :

- Many gastric ulcers cause no apparent clinical signs.
- Signs depend on whether ulceration is complicated (hemorrhage or perforation) or uncomplicated

(A) Uncomplicated cases :

1- Mild intermittent anorexia and abdominal pain.

2- Constipation or diarrhea.

(B) Complicated cases :

(a) With hemorrhage :

1- Melena (Black or tarry faeces) .

2- Pale m.m (Anemia).

3- Sudden death.

b) With perforation :

(1) Severe anorexia.

(2) Intermittent diarrhea (tarry faeces).

(3) Fluctuating fever (Peritonitis).

N.B: Spleen commonly affected with the disease (G.U) .

Diagnosis:

(A) History:

(B) Symptoms:

(C) Lab. Diagnosis : Fecal occult blood analysis.

(D) Instrumentation :

(a)Radiography: Assist diagnosing .

(b)Endoscopy: Most accurate .

Treatment:

(a) Alkalinizing agent :

Neutralize acidity and allow healing e.g. Magnesium hydroxide, carbonate or trisilicate.

N.B: Recently, **omeprazole** inhibit gastric acid produchtion and enhances healing of gastric ulcers in horses.

(b) Coagulants and hematinics :

Against hemorrhage and anemia e.g. vitamin-K, calcium, vitamin-C, thrombin, iron, copper, B12, and folic acid.

N.B: Preparations e.g Homoscon (coagulant) and Fercobsang (hematinics) .

(c) Histamine H-receptor antagonists :

Cimetidine (Zentac) and ranitidine very effective in horses .

(d) Soft food :

To avoid physical irritation.

N.B: Surgical interference is sometimes recommended and gives a satisfactorily results particularly in foals.

* * *

III-GASTRIC DILATATION IN HORSES

Definition:

A syndrome accompanied by signs of abdominal pain and occasionally projectile vomiting.

Etiology :

(A) Acute form:

(a) Following aerophagia while cribbing :

(b) Excessive consumption of water after exercise:

(c) Overeating of highly fermentable.grain ration:

(d) Secondarily to acute intestinal obstruction :

(e) Secandary to acute necrotizing pancreatitis :

(B) Chronic form:

(a) Atony of stomach wall in old or debilitated horses.

(b) External compression of pylorus by tumor mass: e.g. lipoma in horses.

Clinical signs :

(a) Abdominal pains :

Manifested by sweating, rolling and kicking at belly.

(b) Dog sitting posture:

Affected horses sitting on haunches.

(c) Projectile vomiting :

Manifested by little effort- associated regurgitation.

(d)Dehydration signs :

Manifested by sunken eyes and inelastic skil.

(e) Alkalosis signs :

Manifested by tremors, tetany and rapid respiration.

N.B:

Laminits may occur i n cases associated with over eating of concentrates.

Treatment :

Treatment is palliative only :

1- Empty stomach contents (Air, Water, Food) by passage of nasogastric tube

2- Relax pylorus sphincture by local anaesthatic e.g: Lignocaine orally.

3- Give strychnine against stomach wall atony .

4- Supportive I/V fluids and electrolytes therapy.

* * *

IV-GASTRIC RUPTURE IN HORSES

Definition:

Serious complication of sever or untreated gastric distention.

Etiology :

1- Complication of untreated stomach distention.

2- Nasogastric intubation of a distended stomach.

3- Severe overdistention of stomach during falling .

Clinical signs :

1- Sudden relief of abdominal pain.

2-Followed by anxiety and profuse sweating

3- Followed by profound tachycardia and cyanotic m.m. rapid deterioration.

4- Followed by death from hypovolemic shock (Severe haemoconcen tration).

Treatment :

Unsuccessful.

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HORSES INTESTINAL DISEASES
I-ACUTE INTESTINAL OSTRUCTION

Intestine :

(A) Small intestine :

Dudenum -- Juenum – Ileium

(B) Large intestine :

Cecum – large colon – small colon

Etiology :

3 common groups of causes:

1- physical obstruction to the lumen of intestine. (Luminal blockage).
2- physical obstruction to the lumen of intestine plus infarction of the affected section.
3- Functional obstruction with no passage of contents but with the lumen still patent (paralytic ileus).

(A) Luminal blockage

1- Blockage of lumen intestine by foreign bodies, strings, tail hair, phytobezoars ingested with licking-chewing habit.
2- Impaction of ileocecal valve by fine dry indigestible fiber.
3- Impaction of large bowel by enteroliths

(B) Intestinal accidents

1- Intussusception of ileum into cecum or of cecum into cecum or colon into colon.
2- Mesenteric torsion of small intestine (volvulus)
3- Strangulation of an inguinal hernia (in stallions),of an umbilical hernia (in young horses) and of diaphragmatic hernia.

(C) Paralytic ileus

The syndrome of paralytic ileus (functional stasis of intestine) occurs due to :

1- Distention of intestine for periods of more than several days.

2-Acute diffuse peritonitis to the intestines during surgical operations (paralytic ileus is the commonest cause of postoperative fatality cases of surgical colic).

3- In peracute enteritis such as colitis-X

4- In lactation tetany in mares .

Pathogenesis:

Intestinal distention :

C.V. effect- dehydration – Abdominal pain.

1- In severe intestinal obstruction, acute shock due to distention of the bowel which causes reflex cardiovascular effects and peripheral circulatory failure and colla

2- In less severe cases less of fluids and electrolytes and dehydration are the important mechanisms because the fluid and accompanying electrolytes are secreted into the lumen of intestine in response to distention above the obstruction.

3- Distention is responsible foe abdominal pain observed.

N.B :

- Obstruction of upper intestine (duodenum) is most lethal.
- Obstruction of large intestine, may lead to autointoxication due to accumulation of faeces.

Clinical signs :

1- Immediate onset of severe abdominal pain (due to distension at obstruction site) which accompanied by sweating, increased pulse and respiratory rates and tachycardia.

2- Increased pulse and respiratory rate.

3- After 12-24 h of obstruction, the distended intestinal loop can be palpated through the rectum as the defecation ceases and the rectum becomes empty and sticky to touch.

4- After 24 h, dehydration starts and reflux filling of the stomach occurs, which may lead to vomition.

5- Rupture usually occurs at about 48 h.

Diagnosis :

I- History.

II- Clinical signs.

III- Laboratory diagnosis.

Laboratory diagnosis not used for diagnosis of intestinal obstruction, but only useful in assessing its severity.

1- Haemoconcentration (due to dehydration)

2- Leukopenia and neutropenia (due to devitalization of infracted intestine, followed by necrosis).

3- Increase in blood urea nitrogen (due to hypovolemia).

Treatment:

1-Supportive treatment includes :

a)Sedation of the animal.

b)Adm. of antibiotics to control bacterial growth.

c)Electrolytes to overcome hypovolemia, dehydration and shock.

2-Immediate surgical removal of the obstruction.

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II-ENTERITIS

(including enteropathy, malabsorption and Diarrhea)

Definition :

Enteritis means inflammation Of intestinal mucosa resulting in malabsorption and diarrhea and a varying degrees of dehydration and disturbance in acid-base balance.

Etiology :

There are many causes of enteritis or malabsorption in equines, as shown in the following table.

Table (1) Epidemiological and clinical features of disease of horses in which diarrhea is a significant clinical finding.

Etiology	Age and class of animal affected	Major clinical finding
a) physical: 1-Sand colic	Horses grazing on sand pasture or consuming feed contain excessive sand . Usually follows prolonged	Acute or chronic diarrhea, impaction of large colon and faeces contain sand.
2- Stress- induced	Surgical anesthesia	Acute profuse watery diarrhea, severe dehydration and death in 24-48 hrs.
B) Viruses: Rota, corona and adeno – viruses	Newborn foals	Profuse watery diarrhea at few days of age.
Bacteria 1-Sallmonellosis 2- Clostridiosis (cl-perfringens type b and c 3- Corynebacterium equi 4- Actinobacillus	Any age Any age Young foals Newborn foals	Acute profuse foul smelling diarrhea and severe dehydration. Acute profuse watery foul smelling diarrhea and death in 24 hrs. Diarrhea associated with pneumonia Diarrhea and death with 24
e)Parasites 1- strongylus sp. 2- trichonema sp. 3- Ascaris sp.	Usually affect horses over 6 months of age	Acute, subacute or chronic diarrhea according to the degree of infestation.
F) tumors		Chronic diarrhea, progressive loss

Lymphosarcome	Sings horse affected	of weight no response to treatment
G) Miscellaneous (or unknown etiology)		
1- Colitis-X	Single horse affected, usually mature one but may affect yearling may be stress induced.	Peracute profuse watery diarrhea, rapid collapse and death. Poor response the therapy.
2- Granulomatus enteritis	Single horse affected, usually mature one.	Diarrhea not a major clinical finding. Chronic weight loss .
3- tetracycline induced enteritis	Horses treated with 10 times usual dose of tetracycline.	Acute profuse watery diarrhea severe deterioration Death 24-48 hr.
4- Foal heat diarrhea	Foals at 7-10 days at age coinciding with mare's first post partum estrus	Mild diarrhea lasting 1-4 hose. Minimal systemic effects. No specific treatment required.

Clinical findings :

A) Acute enteritis

a) Diarrhea : Faeces are :

1- Soft or fluid consistency.

2-Color other than normal.

3- Odour other unpleasant.

4- May contain blood mucous fibrinous cast or foreign material such as sand.

5- Soil the perineum and tail.

b) Dehydration :

1- Dehydration is usually evident by 10-12 hours following the onset of acute enteritis and clinically obvious by 18-24 hours.

2- The degree of dehydration can be best assessed by tinting the skin of the upper eyelid or neck and determining the time taken for the skin fold to return to normal.

c)Severe abdominal pain :

Acute enteritis usually causes severe abdominal pain manifested by rolling and kicking at the abdomen.

d)Changes in heart rate:

Tachycardia or bradycardia and arrhythmia depending on the degree of acidosis and electrolytes imbalance.

(e) Auscultation of abdomen :

1- In early stages of acute enteritis, auscultation usually reveals sound of increased and fluid-rushing sounds.

2- In later stages, there may be paralytic sounds (no sounds) with only fluid and gas tinkling sounds.

(f) Systemic reactions :

Septicemia, toxemia and fever are common in infectious enteritis.

(g) Peripheral circulatory collapse:

Occurs commonly in peracute and acute enteritis.

N.B :

Malabsorption syndrome commonly referred to granulomatous enteritis and lymphosarcoma of intestine of horses that causing chronic anorexia and progressive weight loss, usually without clinical evidence of diarrhea and slight abdominal pain.

Diagnosis :

I- History.

II- Clinical signs .

III- Laboratory diagnosis.

a) Faecal examined to determine the presence of causative parasites, fungi, bacteria, viruses ...etc. as described under the specific diseases.

b) Blood analysis usually revealed:

1- Haemoconcentration.
2- Metabolic acidosis.
3- Hyponatremia, hypochloremia, and decrease bicarbonate. (hyperkalemia is possible in severe acidosis due to compensatory movement of K^+ from intracellular space to blood)
4- Increase BUN due to inadequate perfusion associated with the dehydration and circulatory failure.

Treatment :

The principles of treatment of enteritis are

1-Temporary withdrawal of the diet if necessary.

2-Removal of the causative agent (Anthelmentics against parasitic enteritis antibacterial against bacterial enteritis, non specific symptomatic treatment for viral enteritis..... etc

3-Replacement of lost fluids and electrolytes.

4- Intestinal protectants and adsorbents.

5-Use antidiarrheal drugs i.g. drugs inhibit secretion and control intestinal hypermotility of necessary.

A) Temporary withdrawal withdrawal of the diet:

1- In foals, temporary withdrawal of feed by muzzling them for 24 hours.
2- Mature horse affected with diarrhea should not have access to any feed for at least 24 hours.

(B) Antibacterials:

1- The use of antibacterials either orally or parenterally, or by both routes simultaneously.
2- Parenteral preparations are indicated in animals with acute diarrhea, toxemia and fever (many antibacterials when given parenterally are excreted by the liver into lumen of the intestine).

3- Oral preparations may be sufficient in cases of subacute diarrhea with minimal systemic effects.

N.B:

-Oral antibacterial preparations should not be used for more than 3 days to avoid superinfection.

-Type and doses of antibacterial commonly used in bacterial enteritis are described under each disease.

(C) Fluids and electrolytes:

To correct the 3 major abnormalities of dehydration, acidosis and electrolyte deficit.

-When severe acidosis is suspected, a 5% hypertonic soln. of bicarbonate is given i/v at a rate of 5-7 ml/kg B.w at a speed of 100ml/ minute.

-Administration of electrolyte solution in quantities necessary to correct the dehydration. :

(a) In severe dehydration (equivalent 10% of B.W), large amount of fluids are necessary at rate of 100-150 ml/kg B.W per 24 hours I/V and I gm of Kcl mast be added for each litre of fluid to overcome hypokalaemia. Used successfully to correct dehydration.

(b) In animals which are not severely dehydrated, oral route can also be used successfully to correct dehydration.

(c) Intestinal protectants and adsorbants :

Kaolin and pectin mixtures are used widely to coat the intestinal mucosa, inhibit secretions and increase the bulk of faeces in horses with enteritis

(e) Antidiarrheal drugs :

Antisecretory drugs for treatment of diarrhea due to hypersecretory activity. For example: atropine sulphate, chlorpromazine Hcl and prostaglandin inhibitors.

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III-Colitis –X

Definition :

Highly fatal acute disease of horses characterized by sudden onset of peracute enteritis manifested by profuse watery diarrhea and rapid development of hypovolemic shock.

Etiology :

The exact cause is unknown, but 2 causes are suggested:

1- Endotoxic shock due to a bacterial toxin.

2- Exhaustion shock due to adrenal corticosteroids hypersecretion.

Clinical signs :

1-Sudden depression.

2- patchy sweating and skin feels cold and clammy.

3-Very sudden onset of enteritis and profuse watery diarrhea.

4- Dehydration and abdominal pain.

5- Muddy discolored mm and dilated pupil.

6- Very fast pulse and elevated temperature (39. 5°C) but soon falls to subnormal before death, which may occur within 4 hours or in less acute cases within 24-48 hours.

Diagnosis :

1-History.

II- Clinical signs.

III- Lab. Diagnosis.

1- Haemoconcentration (PCV may as high as 70%).

2- Severe hypokalemia (serum K^+ may as low as 1.5 mmol/L. while nomal 2.5- 3.7.

3- Marked blood acidosis.

4- Elevation of BUN (over 60 mg%).

5- Leukopenia may be present.

Treatment :

No treatment appears to have any effect on the course of the disease, but the following may be used.

1- Fluids equivalent to at least 10% or even 15% of B.W. e.g.: 40-60 litre/ 400 kg horse) given I/V using indwelling catheter.

2-Massive I/V corticosteroids e.g Dexamethazone 10gm daily for 5 days..

3-Antihistamines e.g I/M or S/C 0.4-0.8 mg/kg B.wt.

4- Broad spectrum antibiotics.

* * *

IV-EQUINE COLIC

Definition :

It is syndrome caused by diseases of alimentary tract characterized by acute, subacute or chronic abdominal pain.

Etiology and types of colic :

(a)Acute colic

Acute colic with severe pain may be caused by:

1-Acute gastric dilataion resulting engorgement with grain.

2-Impaction of the ileocecal valve resulting from feeding finely chopped indigestible roughage..

3-Impaction of the small colon with foreign materials.

4-Accumulation of gas (Flatulent colic) due to ingestion.

5-Intestinal accidents including volvulus; intussuscepsion and strangulation (MECHANICAL COLIC).

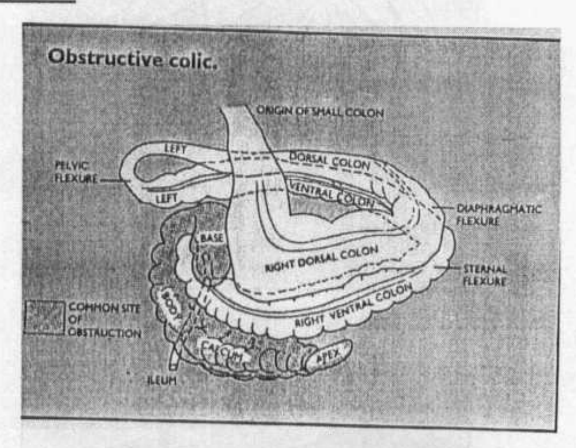
6- Enteritis, especially that caused by the ingestion of sand (SAND COLIC)

7- Hemorrhage into intestinal well as occurs in purpura haemorrhagica and anthrax.

(B) Subacute Colic

Subacute colic includes 2 common forms of the disease.

(a)obstructive çolic:



caused by impaction of the cecum or colon (pelvic flexure) with :

1- Undigested fiber.

2- Dry, firm mass of ingesta.

(b) Spasmodic colic :

Caused by increased gut motility (violent irregular peristaltic movement) and usually following.

1-Periods of excitement.

2- Drinking of cold water after exercise.

3-As a consequence of severe hunger.

4- Sudden change of food.

5- irritation of the gut by unsuitable food stuffs.

C) Chronic Colic

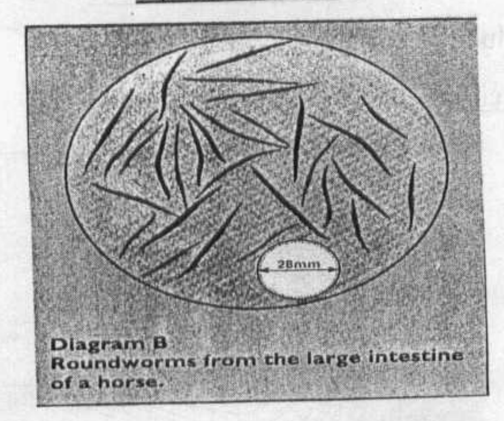
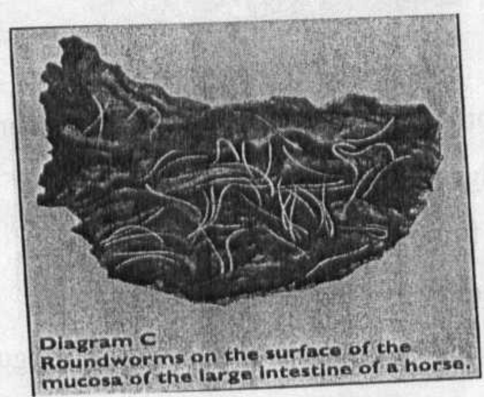


Diagram B
Roundworms from the large intestine of a horse.


Diagram C
Roundworms on the surface of the mucosa of the large intestine of a horse.

1- Verminous aneurysm (Recurrent colic) caused by strongylus vulgaris larvae (verminous thromboembolic colic)
2- old age.
3- Debility.
4- poor teeth.
5- Dietetics error (feeding indigestible roughage).
6- 5-Phytobezoars.
7- Enteroliths .

Clinical findings :

- The clinical signs in different types colic are much the same, varying only in the severity
- Pain observed being almost continuous in acute cases and intermittent in subacute cases. The following manifestation of pain may be observed:

1- Restlessness manifested by kicking the belly and rolling.

2-Looking at the flank (***FLANK WATCHING***) is a common sign especially, in cecal tympany and impaction.



a photo showing flank watching in case of tympany

3- Lie down carefully and get up slowly, especially in flatulent colic.

4- Often adopting a dog-sitting posture especially in acute gastric dilataion.

5- Affected horses may adopt other abnormal postures, including saw horse attitude and lying on the back.



a photo showing a horse laying on back with paddling with legs due to colic	a photo showing a horse with lateral recumbency and paddling with legs due to colic

6- In the most severe cases there is

- profuse, patchy sweating
- Sobbing respiration
- Signs of shock including rapid pulse and clammy skin
- The horses movements are so violent that self physical injury occurs.

7- Auscultation of the abdomen is helpful in diagnosis.

- In flatulent colic, there are high – pitched "gassy pings"

- In spasmodic, there are loud gut sound (continuous borborygmi).
- in obstructive colic, the normal sounds are decreased or absent.

8- Rectal examination is essential in diagnosis :

- In flatulent colic, there is gaseous distention of intestinal loops.
- No abnormalities are detectable in spasmodic colic.
- Impaction of cecum or colon are readily palpable.
- In impaction of ileocecal valve, a cylindrical mass in the terminal part of ileum high up in the right flank.
- In case of verminous colic, the obstructed a.v. may be palpable and slack, distended loops of intestine can be found.
- Small intestinal accidents are characterized by fluid-filled loops of gut of appropriate size.
- Hernias into the inguinal canal are best palpated per rectum, as the intestine may not protrude as far as the scrotum .

9- Projectile vomiting of evill-smelling, green fluid usually result from an obstruction of the intestine at any level from pylorous to the ileocecal valve.

Treatment :

1- relief the pain by analgesics e.g Meperidine 2-4 mg/kg B.wt. S/C or chloral hydrate 15-30 gm (by stomach tube) or Buscopan. In mild colic attacks tranquilizers can be used e.g. Acepromazine maleate 2-4 mg/100.lb .B.wt. I/V/ min. or S/C.

2- rectal enema with soap and water may be useful.

3- in impaction colic, mineral oil lubricant or laxative is best treatment (2-4 litres)

4- In sand colic, magnesium sulfate is recommended

Magnesium sulphate	300 gm
Formalin	30 ml
Water	12 litres

Warm to body temperature and given by stomach tube.

N.B:

In both impaction colic and sand colic, these treatments may be followed 12-24 hours later by injection of carbachol 1.2-4ml 1:1,000.

5- In spasmodic colic, a spasmolytic e.g atropine sulphate 15-30 mg/S/C is effective antispasmodic.

6- In flatulent colic the following prescription can used as a single dose by stomach tube RX:

Cresote	6ml
Ammonium carbonate	16ml
Speritous ether initrosi	36ml
Turpentine oil	60 ml
Lin-seed oil	1000 ml

7- In constructive and mechanical colic's laparotomy may be useful.

8-In verminous colic, horses may respond to classical therapy, but may respond to a massive doses of appropriate anthelmintic

Prevention :

In all cases and types of colic, dental attention, advice on feeding and exercise and effective parasitic control are best program for prevention in horses.

▪ SPASMODIC COLIC

Definition:

It is a subacute form of colic caused by hypermotility of intestine and characterized by short attacks of abdominal pain and loud borborgymi sound

Etiology :

1- Period of excitement as preparation for showing or racing.
2- Drinking of cold water after exercise.
3- As sequence of severs hunger.
4- Sudden change of diet.

5- Irritation of the gut by unsuitable foods stuffs.

Pathogenesis :

The hypermotility in spasmodic colic is thought to arise by an increase in the parasympathetic tone under the influence of the causative factors mentioned above.

Clinical findings :

1- Short attacks of abdominal pain.

2- Pain is intermittent the horse rolls. Paws and kicks for a few minutes. Then shakes itself and stands normally for a few minutes until the next attack of pain occurs.

3- intestinal sound are often audible distance from the horse and loud rumbling borborygmi sound are heard on auscultation.

4- The pulse is moderately elevated and there may be some patchy sweating.

5- The signs disappear spontaneously within a few hours.

6- There is no scouring.

Diagnosis :

1- Case history. 2- Clinical signs.

Treatment :

1- Using of atropine sulphate as spasmolytic followed by ½ gallon of mineral oil by nasal tube.

2- Injection of mepridine HCL 2-4mg/kg BW s/c .

3- Novocaine (0.05g/ 100 Ib B.W) injected slowly i.v. may produce immediate relief of pain which lasts for 15-20 minutes.

4- Promazine derivatives have a tranquilizing and spasmolytic effect and followed by a mild purgative appear to be treatment of choice.

THROMBOEMBOLIC COLIC
(VERMINOUS ANEURYSM)

Definition :

It is a chronic recurrent attacks of abdominal pain caused by migration of the larvae of *Strongylus Vulgaris* in the cranial mesentric artery.

Etiology :

1- Migration of the larvae of *Strongylus Vulgaris* into the of the cranial mesentric artery and its branches causing restriction of the blood supply or damage of the nerve supply to the intestine.

2- Horses in pastures are more susceptible to strongylosis even with good worm control.

Pathogenesis :

1- Migration of the larvae of *Strongylus Vulgaris* causes thrombosis in the mesentric vessels. The thrombosis causes restriction of the blood supply to the intestine leading to typical infarction of the bowel without displacement of the nowel.

2-Secondary bacterial infection mainly by streptococcus equuli actinobacillus equuli, or salmonella causing mesenteric abscess, which produces signs similar to those of aneurysm but the temp. is usually elevated and rupture of the abscess may lead to a diffuse peritonitis.

3- Fatal termination may occur due either to peritonitis after rupture of the intestinal wall or toxemia caused by gangrene of the intestinal wall.

Clinical Signs :

1- Severe abdominal pain for 3-4 day with almost complete cessation of defecation.

2- In early stages there may be increased gut sounds to the degree where spasmodic colic suspected while in later stages the intestinal sounds are decreased.

Diagnosis :

1- Case history and clinical signs.

2-Radiographic examination to determine the verminous aneurysm

3- Lab. Examination of faeces reveals.

a- High count of Strongylus eggs.

b- Bloody – stained faeces in case of rupture

4- Rectal findings disclose the following

a- Distended loops of intestine.

b- Tightly stretched mesentry .

c- firm swelling in the root of mesenteric artery

d- Enlargement of the colic and cecal arteries.

Treatment :

1- Anthelmintic such as thiabendazole 500mg/kg B.W. orally

2- Supportive treatment. Fluid therapy

3- I.V. injection of plasma expander dextran 2.5 ml/kg B.W./ day for 3 days followed by further injections at 4- day intervals for a total of 9 injection (has anticoagulant properties)

4- Surgical interference .

▪ SAND COLIC

Definition:

It is an acute type of colic caused by ingestion of sand that cause irritation or obstruction of the GIT.

Etiology and pathogenesis

1- Irritation or obstruction of the gastrointestinal tract is a problem when sand is ingested by horses.
2- Sand is consumed when it becomes mixed with hay fed on the ground, when horses graze short grasses, when they drink from shallow muddy pool,s and when horese become salt deficient.

3- The large colon and in particular the right colons, pelvic flexure and transverse colon are the most common sites of obstruction because they are fermentative areas of reduced flow wheree sand consequently settles and transvere colon become obstructed when masses of accumulated sand become lodged in these narrow segments.

Clinical signs :

1- Sand in the intestine causes both mucosal irritation and mechanical obstruction.

2- The mucosal rritation results in diarrhea, which precede mechanical obsturuction of the bowel by several days.

3- When obstruction becomes distended with gases, fluid and ingesta, pain results from stretching of the bowel wall.

4- The weight of the impacted mass as well as distention of bowel result in twisting or displacement of the impacted mass and necrosis of the bowel wall at the site of the impaction.

5- Perforation of the bowel wall at the site of obstruction may occur. Both displacement and perforation result in pain and development of shock.

Diagnosis :

1- Simple observation of feeding practices and environment will reveal the opportunity for ingestion of sand.

2- History of diarrhea followed in several days by mild colic is indicative of sudden obstruction of the gastrointestinal tract.

3- Abdominal distension and gastric reflux are the results of complete obstruction or displacement or both.

4- Rectal palpation is not conclusive because the impaction occurs most frequently in areas that are not within reach and the weight of the sand caused the bowel to rest on the ventral abdomen.

5- During rectal palpation, samples of faeces should be examined for the presence of sand. Six fecal balls can broken up into a quart pitcher of

water and the sand is allowed to settle. More than a teaspoonful of sand in the container is abnormal.

Treatment :

1- If pain is mild to moderate and can be controlled by analgesics and the horse is still passing stool. Medical therapy is indicated. Medical therapy should include laxatives and fluids to maintain hydration and a diet of high quality roughage. Saline purgatives are preferred as will bring fluid into the bowel prevent desiccation of mass and help passage of sand physiological saline and dextrose must be injected i.v. in case of chock.

2- When the pain is uncontrolled, abdominal distension or bowel distention on rectal palpation is marked and the condition does not respond for medical therapy within 48-72 hours, surgical interference is indicated. Delaying surgical interference will result in necrosis and rupture of the bowel.

* * *

V-INTESTINAL TYMPANY

Intestinal tympany causes distention of the abdomen and severe abdominal pain and is sometimes accompanied by the passage of much flatus.

Etiology :

-All cases of tympany of small intestine are caused by same forms of intestinal obstruction.

-Tympany of large intestine may be primary or secondary

a- Primary large bowel tympany results from the ingestion of large quantities of highly fermentable green feed.

b- Secondary large bowel tympany results from stenosis by constricting fibrous adhesions after castration or in association with verminous aneurysm.

Clinical Signs :

1- Abdominal pain and affected animal may roll and paw.
2- intestinal peristaltic sounds are reduced but fluid may be heard moving in gas-filled intestinal loops. Producing tinkling and metallic sounds.
3- On rectal palpation gas-filled loops of intestine can be felt.
4- Much flatus is passed and the anus may be in state of continuous dilatation.

Treatment :

1- In severe primary cases, trocarization with a long small-caliber intestinal trocar and canula is used. This can be performed though the upper right or left flank descending the maximum distension.
2- Mineral oil 24 liters and antifermentative materials as turpentine oil.
3- In secondary tympany, permanent relief can be obtained only by correction of the obstruction

PERITONITIS

Definition :

Peritonitis means a local or generalized peracute, acute, or chronic inflammation of peritoneum.

Etiology :

1- Rupture of the stomach due to ulceration by larvae of Gastrophilus or Habronema spp. Or any other causes.
2- Rupture of dorsal sac of cecum or colon during foaling.
3- Rupture of rectum during rectal examination (predisposed by inflammation of mucosa)
4- Cecal perforation due to heavy infestation by Anaplocephala tapeworms.

5- Administration of non-steroidal anti-inflammatory drugs causing cecal stasis and dilation and eventually perforations.

6-Chemical peritonitis result from presence of blood, chyle, urine, bile or pancreatic juice released into peritoneal cavity.

7-Extension from retroperitoneal infection e.g. streptococcus equi after an attack of strangles.

8-Actinobacillus equuli by unknown mean.

9-Traumatic perforation from the exterior of abdominal wall.

10-Bacterial contamination following surgical intervention (laparotomy), trocarization of cecum and peritoneum .

Clinical signs :

(a) Peracute diffuse peritonitis:

In mares the rupture of dorsal sac of cecum occur during foaling there are moderate abdominal pain followed by shock (due to toxemia and bactremia) and death after 6-18 hours of rupture.

(b) Acute diffuse peritonitis:

1- Complete anorexia.

2- Toxaemia and fever.

3- Faeces may be completely absent for up to 3 days.

4- Alimentary tract stasis evidenced by absence of gut sounds on auscultation (paralytic ileus).

5- Abdominal pain (colic) including flank watching, kicking at the belly and rolling.

6- Palpation of abdominal wall feel tenderness (stiffness).

C) Chronic peritonitis :

a- Horses with chronic peritonitis usually have a history of ill-thrift for a period of several weeks.

b- Clinical signs are.:

1- Severe weight loss.

2- Intermittent episodes of abdominal pain.

3- Reduced or absent of gut sounds.

Treatment :

a- Specific treatment for the primary cause.

b- Non specific treatment includes.:

1- Administration of antibacterial drugs e.g. broad spectrum antibiotics directly into peritoneal cavity.

2- Treatment of toxaemia.

* * *

HORSES LIVER DISEASES

I-HEPATITS

Definition :

Hepatitis means diffuse acute or chronic inflammation of the liver:

- Diffuse chronic inflammation means liver cirrhosis.
- Clinically the syndrome caused by cirrhosis of the liver is the same as that caused by acute hepatitis, the only difference being that the onset of the disease is slower and less acute than in hepatitis.

Etiology :

(A) Toxic hepatitis

a- Inorganic poisons e.g. phosphorous, copper, selenium... etc.

b-Organic poisons e.g. carbon tetrachloride, hexachlorethane, gossy pol, creasol, coal tar...etc...

c-Poisonous plant, e.g. sore weeds, fungi, algae... etc.

B) Infectious hepatitis

1- Infectious equine anemia.

2- Viral rhinopneumonitis.

3- Severe cases of equine viral arteritis.

4- Salmonellosis.

5- Bacillus piliformis (tyzzers disease foals)

6- Systemic mycosis e.g histoplasmosis.

7- Migrating larvae of Ascaris sp.

Clinical findings:

(A) Acute hepatitis:

1- Anorexia.

2- Constipation intermittent with attacks of diarrhea.

3- Faeces lighter than normal .

4- Subacute abdominal pain manifested by arching of back.

5- Nervous signs vary from ataxia and lethargy with "yawning" or coma to hyperexcitability with muscle tremor, mania, including aggressive behaviour, and convulsions .

6- Jaundice and oedema may or may not be present.

7- Photosensitization in animals fed green feed only when exposed to sunlight.

8- A tendency to be more feely than normal may be observed.

(B) Chronic hepatitis (cirrhosis):

- The signs are similar to those of acute hepatitis but develop more slowly, and persist for longer periods; often months.
- Ascitis and the dummy syndrome are more common in chronic hepatitis

N.B:

Dummy syndrome is a characterized syndrome observed in animals affected with hepatitis especially chronic one (cirrhosis) in which affected animals push with the head, does not respond to normal stimuli, and may be blind.

Diagnosis :

1- History

II- Clinical signs.

III- Lab. Diagnosis.

Laboratory examination of blood (liver function tests) and urine samples and liver biopsy specimens.

Treatment :

1-Avoid protein and protein hydrolysate to avoid danger of ammonia in toxication due to failure of liver detoxification mechanism and subsequent nervous signs.

2- The diet should be high in carbohydrate and calcium and low in protein and fat.

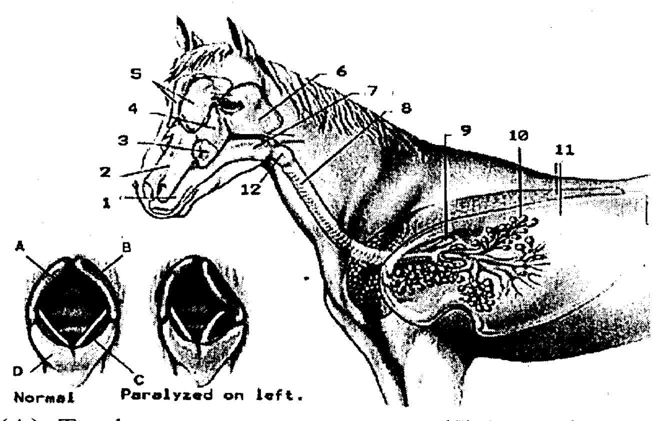
3- Oral administration of broad-spectrum antibiotics (neomycin) to control protein putrifaction .

4- Periodic injections of water-soluble vitamin particulary vitamin B complex.

N.B:

Hepatic cirrhosis is considered to be a final stage and treatment is not usually undertaken.

DISEASES OF RESPIRATORY SYSTEM



(A). Trachea
(B). Cartilage
(C). Vocal cord
(D). Epiglottis
(1) Buccal cavity
(2) Nasal Cavity (open to pharynx)
(3) Inferior maxillary sinus
(4) Superior maxillary sinus
(5) Frontal sinuses
(6) Guttural pouch
(7) Pharynx
(8) Trachea
(9) Bronchus
(10) Alveolus
(11) Lungs
(12) Larynx

EPISTAXIS (NOSE BLEED)

- Epistaxis mean bleeding from the nostrils regardless of origin of the hemorrhage.
- hemoptysis means coughing of blood with hemorrhage usually originating from the lungs.

Etiology :

a- Epistaxis occurs commonly in the horses and may be due to lesions in nasal cavity, nasopharynx, guttural pouch or lungs.

b- Hemorrhage lesions of nasal cavity, nasopharynx and guttural pouch usually cause unilateral epistaxis, while blood originating from the

lungs is discharged from both nostrils and not the mouth because of the long soft palate.

N.B:

Blood originating from the lungs not foamy as was previously thought because the horizontal position of the major bronchi allows blood to flow out freely without being coughed-up and made foam. Froth is usually the result of pulmonary edema in which case it is very fine pink stable froth.

c- The commonest cause of epistaxis in horses are:

1- External trauma.

2- Foreign bodies in nasal cavities.

3- Neoplasms (erosion of m.m. of nasal cavity).

4- Mycotic ulceration of B.V. in the wall of guttural pouch around the guttural pouch.

5- Encapsulated hematoma in nasal cavity.

6- Hemorrhage polyps of m.m. of nasal cavity or paranasal sinuses.

7- Erosion of the nasal mucosa in glanders.

8- Internal trauma due to passage of nasal tube or endoscope.

9- Congestive heart failure (mild epistaxis).

10- Purpura hemorrhagica.

11- Thrombocytopenia (generalized bleeding).

12- Idiopathic in exercise-induced epistaxis in race horses (bleeders).

Diagnosis:

I- History

II- Clinical signs.

III) Exam of nasal cavities **:**

1- Visually with the aid of strong pointed source of light through external nares.

2- with the use of flexible fiberoptic endoscope will permit thorough exam. Of the nasal cavities, nasopharynx, guttural pouch larynx, trachea and major bronchi.

Treatment :

a- Treat the cause, if it is known.

b- when the cause is obscure, treat symptomatically as follow:

1- Cold packs over the nose and forehead.

2- Tampon or gauze soaked in adrenaline inserted in zigzag manner into affected nostril (but not in both nostrils).

3- vasoconstrictor drugs e.g. thrombin solution spray on bleeding surface immediately, or diacynon injection.

4- Calcium preparations e.g. calcium gluconate 20% 500 ml. S/C or slow I/V.

5- Vitamin – K injection (synkavit = synthetic – vitamin – K)

6- Blood transfusion in severs cases.

7- Hematinics e.g. iron preparations.

N.B:

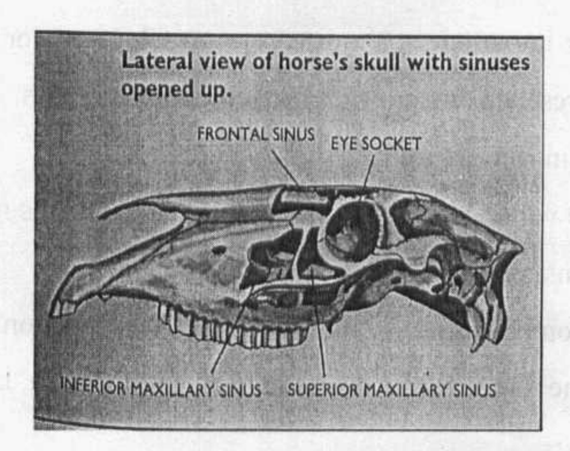
Avoid injection of adrenaline as it increasing bleeding by rising of blood pressure.

RHINITIS (CORYZA)

Definition :

Rhinitis means inflammation of m.m of nasal cavity which may be acute or chronic, unilateral or bilateral, characterized by sneezing, wheezing and stertor during inspiration and nasal discharge which may be serous, mucoid; purulent or blood – tinged in consistency depending on the cause.

Etiology :



(A) Rhinitis is present as a lesion in the following conditions:

1- Dusty stable.

2- Inhalation of irritating chemicals.

3- Nasal foreign bodies .

4- Extension of alveolar infection.

5- Nasal or sinus tumors.

(B) Rhinitis is present as a lesion in the following diseases:

1- Strangles.

2- Glanders.

3- Epizootic lymphangitis

4- Equine rhinovirus.

5- Equine Viral rhinopeumonitis (herpes virus)

6- Influenza viruses

7- Parainfluenza viruses.

8- Adenovirus.

9- Reovirus

10- Hoppengarten cough virus

Clinical signs :

-Rhinitis is of minor importance as a disease proceed. Its major importance is indication of the presence of some specific infections diseases.

- The cardinal signs in rhinitis are :

1- Nasal discharge which is usually serous but soon becomes mucoid, and in bacterial infections, purulent.

2- Erythema, erosion or ulceration may be visible on inspection of nasal m.m

3- Sneezing in the early acute stages which followed in later stages by snorting.

4- Associated L.n. may show signs of inflammation.

N.B:

Chronic unilateral purulent nasal discharge lasting several weeks or months may suggest nasal mycosis (aspergillosis, or Cryptococcus) Diagnosis:

(1) History

(II) clinical signs

(III) Lab. Diagnosis

Exam. Of nasal swabs of scraping for bacteria, inclusion bodies or fungi may aid in diagnosis .

(IV) Endoscopic examination :

Endoscopic examination using a flexible fiberoptic endoscope is very useful for the visual inspection of lesions affecting the nasal mucosae which are not visible externally.

Treatment :

1- specific treatment to control causative agents as described under the specific diseases and causes.

2- remove the thick tenacious exudates which causing nasal obstruction using warm boric acid, then irrigate the nasal cavity with saline or mixture of saline and antibiotics to provide symptomatic relief and minimize secondary bacterial rhinitis .

3- Spray – up nasal decongestant into the nostrils to provide some relief e.g.. Delta rhinol spray.

4- Culture and sensitivity testing to assist the selection of suitable antibacterial agents, which may be helpful in controlling secondary bacterial infection.

Diseases Of The Guttural pouch

1- Guttural pouch mycosis:

Occur horses as result of an infection entering the Eustachian tube via its pharyngeal orifice (e.g. Aspergillus sup)

At least 4 different sequels to guttural pouch mycosis may result:

1- Erosion of internal carotid artery with the sudden development of profuse epistaxis, usually in a horse at rest.

2- Thrombus development in the internal carotid artery with emboli being shed to locate, unilaterally in brain resulting in blindness and ataxia.

3- Mycotic encephalitis.

4- Inflammation of the cranial nerves anatomically close to the wall of the guttural pouch.

The common effects of damage to these nerves are:

a-Dysphagia due to involvement of pharyngeal branches of hypoglossal and vagal nerves.

b-laryngeal hemiplagia(roaring disease) due to involvement of laryngeal branches of vagus.

c-Facial paresis due to involvement of facial nerve.

d-Horner's syndrome (ipsilateral facial sweating and hyperthermia, smaller palpebral fissure, mild miosis) due to involvement of sympathetic neurons.

II- Guttural pouch empyemia :

Accumulation of pus in the guttural pouch.

The causes of guttural pouch empyemia or sepsis usually:

1-Extension frôm pharyngitis (especially streptococcus infection)

2-Rupture of an abscess of retro-or supra pharyngeal lymphnode.

-The clinical signs of guttural pouch empyemia are:

1- Chronic toxaemia (manifested by pyrexia)

2- distension of one or both pouches.

3- pain on swallowing and dysphagia

4- Coughing

5-Intermittent mucopurulent or purulent nasal discharge especially when animal lowers its head.

III- Guttural pouch tympany:

1- The disease is limited in occurrence to foals a few days old.

2-The disease occur due to congenital defect of closure of the Eustachian tube that a lows the pouch to fill with air and distended obviously and to an enormous size.

Diagnosis:

1- History

II- clinical signs

III- Endoscopic exam of pharynx.

Treatment :

1-Catheterization and irrigation of the guttural pouches with the following.

a) Iodine solution in guttural pouch mycosis.

b) Antibiotics solution in guttural pouch empyemia.

Pencillin K 5.000.000 I.U.

Dihydrostroptomycine 2.0 gm.

Isotonic salt solution 100 ml.

Flush into guttural pouches daily for 7-10 days.

1- palliative treatment by systemic administration of :

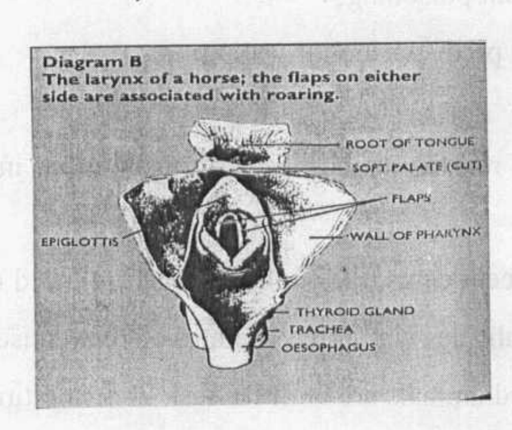
(a) Iodides to control mycosis or 15- minutes application of 30 ml nystain (mycostatin oral suspension).

(b) Antibiotics paranterally to control empyemia.

3- Surgical drainage for unresponsive infections.

DISEASE OF LARYNX

Roaring disease (Laryngeal hemiplegia)



Definition :

Roaring disease is a chronic, unilateral or occasionally bilateral paralysis of the intrinsic muscles of the larynx causing audible inspiratory dyspnea.

N. B:

- Paralysis occurs on the left side in 92% of the recorded cases, on the right side in 6% and on both side in 2%, this observation has led to theory that the disease is related in some way to constant irritation produced by pulsations of aorta as the left recurrent laryngeal nerve passes around it.

Etiology:

a- Degeneration or injury of one or both or both of the recurrent laryngeal nerves. As a result, the arytenoids cartilages and the corresponding vocal cords fail to rotate outward on inspiration, causing reduction in the size of the lumen of the larynx with consequent inspiratory dyspnea.

b- The causes of degeneration or injury of the nerve may be:

1- Accidental injury to the nerve by over extension of head.

2- Previous injection of irritating substances.

3- Previous infectious disease e.g. Strangles.

4- Guttural pouch mycosis.

5- Debilitating diseases.

6- Lead poisoning.

7- Certain plant poisoning.

8- Hereditary predisposition.

Clinical findings :

1- Whistling or roaring sound heard on inspiration, in severe or advance cases.

2- In mild or recent cases, the sound may be produced only after strenuous exercise and subsides within few minutes after exercise stopped.

3- Badly affected animals are unfit for fast work and tire quickly as a result of the dyspnea.

4- Experienced clinicians may be able to detect a pit between the arytenoid and thyroid cartilages on laryngeal palpation.

Diganosis:

(I) History

(II) Clinical signs

(III) Endoscopic examination:

Endoscopic examination of the larynx confirmed the diagnosis.

Treatment:

1- Spontaneous remissions rare.

2- Surgical correction is:

a- Extirpation of the laryngeal ventricle (ventriculectomy) is successful in restoring about 60% of the affected horses.

b- Surgical retraction of the cartilages has proven effective in cases that have not respond to ventriculectomy.

LARYNGITIS, TRACHITIS AND BRONCHITIS

(Upper respiratory tract affection)

Etiology:

1- Equine viral influenza. 2- Equine viral arteritis.

3- Equine herpesvirus. 4- Strangles (streptococcus equi)

Clinical findings:

Coughing and inspiratory dyspnea are the two common clinical signs.

1- In the early acute stages, the cough is usually dry non-productive and is easily induced by grasping the larynx and trachea.

In the chronic stages, the cough may less frequent distressing and usually dry and harsh.

N.B:

1-Moist cough and thick mucous, flackes of blood and fibrin may be coughed if the lesions cause much exudation or ulceration of the mucosa.

2- Inspiratory dyspnea varies with the degree obstruction and is usually accompanied by a loud stridor and harsh breath sounds on each inspiration (these are best heard over the trachea) .

Diagnosis:

(I) History

(II) Clinical examination.

(III) Endoscopic examination:

Examination of upper respiratory tract with fiberoptic endoscope

Treatment:

1- Rest and avoidance of exposure to inclement weather may resolve spontaneously the common viral infection.

2- Secondary bacterial complication must be treated with the appropriate antibacterial agent (antibiotics or sulfonamides)

3- A combination of corticosteroid (Dexamethazone) and antibiotics therapy is of value in chronic cases in horses.

N.B:

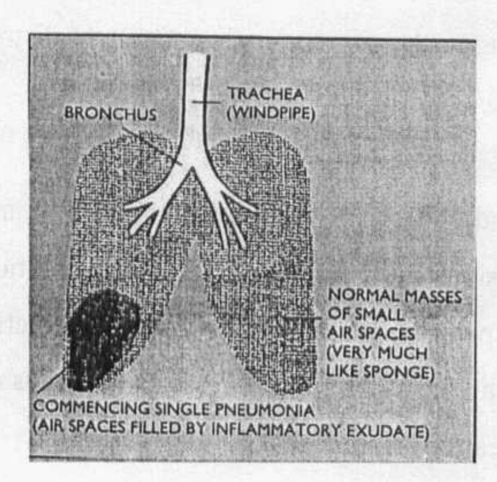
- Animals with severe lesions and marked inspiratory dyspnea may require a tracheotomy and insertion of a tracheotomy tube for several days until the lesion heals.

PNEUMONIA

Definition :

Pneumonia is inflammation of pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleurisy

Etiology :



(A) predisposing factors:

predisposing factors weaken the defense mechanisms of the animals e.g.

1- Inclement weather. 2- poor ventilated housing.

3- stress of transportation. 4- Stress of malnutrition.

(B) infectious causes:

a- Viral pneumonia

1- Adenovirus pneumonia (in immune – deficient foals).

2- Equine herpes virus (older foals)

3- Equine influenza virus (older foals)

4- Equine viral arteritis (adult horses).

5- Equine viral rhinopneumonitis (adult horses).

b- Bacterial pneumonia :

1- E. coli streptococcus equi, Actinobacillus equuli which causes newborn foal septicemia.

2- Corynebacterium equi (older foals)

3- Strangles

4- Glanders

5- Pleuropneumonia due to anaerobic bacteria.

C- Mycotic pneumonia:

1- Epizootic Iymphangtitis

2- pneumonic aspergillosis

d- Verminous pneumonia:

1-Dictyocaulus arnifeldi.

2- Parascaris equorum.

Clinical findings :

1- rapid, shallow respiration is the cardinal sign of early pneumonia.

- Dyspnea occurring in the later stages when much of the lung tissue is non – functional

2- Cough which is:

- Dry, frequent, hacking cough in interstitial pneumonia.
- Moist, painful cough in bronchopneumonia.

3- Cyanosis: Not a common sign, Occurs only when large areas of the lung are affected.

4- Nasal discharge: May or may not present depending upon the amount of exudates present in bronchioles and whether or not, there is accompanying inflammation of the upper respiratory tract.

5- Abnormal breath odour: Decay – putrid – Decay when there is a large accumulation of inspissated pus.

- Putrid when pulmonary gangrene is present.

6-auscultation of the lunges – in early – crackles – In early congestive stage of bronchopneumonia and interstitial pneumonia there is in creased breath sound (bronchial sound)

-Crackles (moist rales) develop in bronchopneumonia as bronchiolar exudation increases.

N.B:

Consolidation also causes increased audibility of the heart sounds. Pleuritic friction rub in early stages when pleurisy. Is also present and muffing of bronchial sounds in the late exudative stages.

7- in acute bacterial bronchopneumonia, there is toxemia, fever, anorexia, depression and tachycardia.

Diagnosis :

(I) History

(II) Clinical

(III) Endoscopic examination

(IV) Nasal swabs

Treatment :

1- Isolation of affected animals (particularly if infectious disease suspected) in warm, well ventilated, draft free place and provide with ample fresh water and light nourishing food (parenteral force – feeding of animal not eat)

2- The choice of antibacterial agent (antibiotic or sulfonamide) based on culture and sensitivity testing.

Drug the choice of antiparasitic agent if verminous pneumonia suspected.

3- The use of corticosteroid as and anti-inflammatory agent e.g. Dexamethazone, Betamethazone.

4- The use of bronchodilator to improve ventilation with a resulting net improvement in oxygen exchange.

The most commonly used agents are aminophylline and theophylline .

Sympathomimetic drugs such as epinephrine is effective but little used because of their – term action.

Diagnosis :

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(II) Clinical signs

(III) Endoscopic examination

(IV) Nasal swabs

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The most commonly used agents are aminophylline and theophylline

Sympathomimetic drugs such as epinephrine is effective but little used because of their short – term action.

The beta-2 adrenergic receptor selective bronchodilators, such ad clenbuterol, a new being used in COPD and exert a very beneficial effect.

5- Antihistaminic e.g. Avil injection or tussivan syrup.

6- the use of execptorants according to type of cough.

- When cough is painful and exhausting and the secretion tenacious, sedative expectorant, such as ammonium or potassium salt, stimulate secretion of protective mucus and lessen coughing
- When cough is soft and bronchial exudates is voluminous, as in bronchopneumonia, stimulant expectorant is more valuable.
- When cough is exhausting and interferes with activity but there is little exudation, anodyne expectorant, such as belladonna, codeine, morphine or heroin is indicated.

ASPIRATION PNEUMONLA

(Drenching pneumonia, Inhalation Pneumonia)

Etiology :

1- Careless drenching or passage of a stomach tube during treatment for other illness.

2- Following vomiting

3- Rupture of pharyngeal abscess.

4- Paralysis obstruction of larynx, pharynx and esophagus result in aspiration of foal of water when attempting to swallow.

Clinical signs :

1-If large quantities of fluid are aspirated death may occur suddenly

2- If small quantities are aspirated, the outcome may depend on the composition of the aspirated, the outcome may depend on the composition of the aspirated material e.g

-With soluble such as chloral hydrate and magnesium sulphate, very rapid absorption from the lung occur.

-With insoluble substances and vomitus, pneumonia with toxaemia result, which is usually fatal in 48-72 hrs.

3- The severity of aspiration pneumonia depend largely upon the bacteria which are introduced, causing in many cases an acute gangrenous pneumonia.

Treatment:

As mentioned before with pneumonia.

Chronic obstructive Pulmonary Disease (COPD)
(Bronchiolitis – Emphysema Complex)

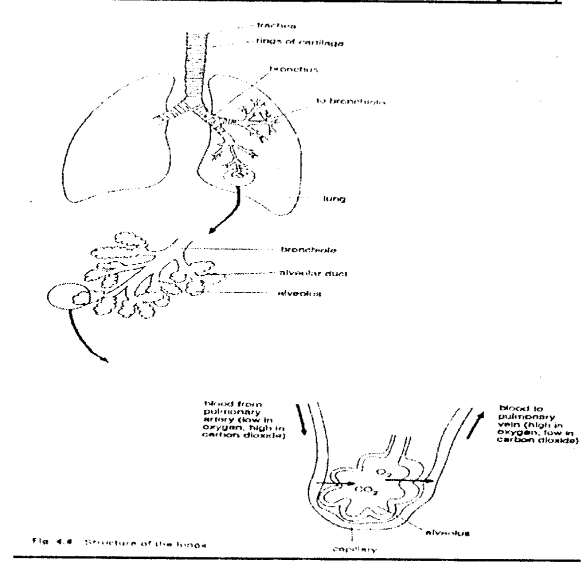


Fig 4.4 Structure of the lungs

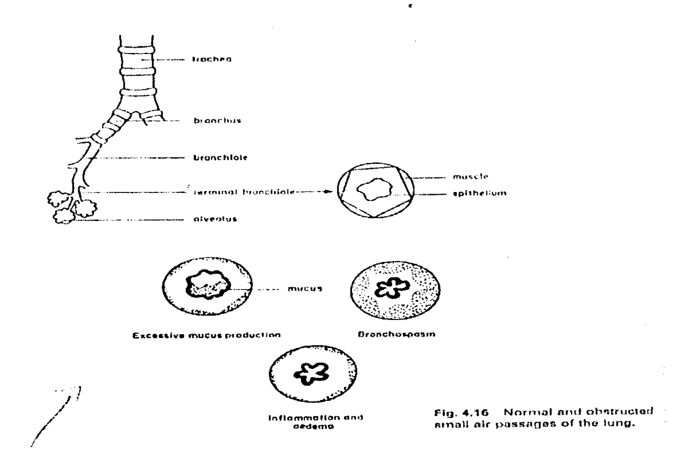


Fig. 4.16 Normal and obstructed small air passages of the lung.

The disease was formerly known as "Heaves", poor man disease, chronic alveolar emphysema , pulmonary emphysema or broken wind

Definition :

- COPD in the horse is characterized clinically by decrease work performance, chronic coughing, abnormal lung sounds and cardiac dysfunction.
- Pathologically, there are varying degree of bronchiolitis and pulmonary emphysema.

N.B:

Pulmonary emphysema is a distension of the lung caused by over distension of the alveoli with rupture of alveolar wall with or without escape of air into interstitial spaces.

Etiology:

- The exact cause of the disease in unknown.
- Epidemiological evidence of the disease suggest the following causes.
  1- Hypersensitivity reaction to "allergens"found in barn dust and moldy and dusty feeds such as, Aspergillus fumigatus and Micropolyspora.
  2- as a sequelle to viral infection of the upper respiratory tract of the horse.
  3- Secondary to bronchopneumonia

Clinical findings :

On clinical examination, the horse is usually bright and alert, the temperature is normal and appetite is usually normal

1- Coughing (single or paroxysm) which becomes more pronounced and wheezing with exercise.

2- intermittent, bilateral nasal discharge which may be serous, mucoid mucopurelent or blood stained.

3- The resting respiratory rate is increased from a normal of 12/min. up to 24-36/min.

4- Expiration is biphasic or more exaggerated (double expiratory effort).

5- During expiration there is normal collapse of the rib cage followed by a clearly visible contraction of the abdominal muscles of flank.

N.B:

In longstanding cases this result in the so-called Heave – line which is a trough which follows along the costal arch (Barrel chest)

6- Auscultation of the lungs reveal wheezing and crakling sounds occur at the end of inspiration and the end expiration (placement of plastic bag over the horse's nostrils for 1 minute will cause the horse to hyperventilate and abnormal sounds are more pronounced)

N.B:

Pleuretic friction rib may be auscultated over the chest

7- Percussion of the thorax may reveal a hyper resonant sound and also revealed an increase in the area of resonance by as much as 1 – 2 intercostal spaces caudally.

8- increasing in heart rate up to 50-60/min in advanced cases of COPD due to pulmonary arterial hypertension.

N.B:

COPD may resulted in ventricular right – sided hypertrophy and congestive heart failure.

Diagnosis:

(I) History:

1- The disease occurs most commonly in adult horses over 5 years of age (6 – 10 years of age)

2- the usual history is that the hás been stabled for several weeks or months and has a chronic cough.

3- The horse may have had an infections respiratory disease 1 or 2 months previously, recovered from the acute illness but began coughing recently.

(II) Clinical findings :

(III) Lab. Diagnosis:

1- Blood gas analysis in COPD reveal PaO_2 below normal and $PaCO_2$ us increased.

2- Pulmonary function test.

3- precipitins against fungi have been identified in serum of affected horses.

4- allergic skin test and antigen inhalation tests have also been used.

5- Cytological examination of tracheobronchial aspirates with COPO reveal excessive mucus and neutrophils.

6- Endoscopic examination of upper respiratory tract.

7- Radiographic examination of thorax may be useful in differentiating COPD form other diseases of the lungs of horses.

Treatment and Control :

There is no specific cure, treatment is palliative.

1- The provision of fresh air, as the horse should be kept permanently in the open air.

2- Avoid exposure of the horse to dust, therefore wood chippings or saw dust should be used for bedding instead of straw.

3- Corticosteroids, such as dexamethazone 25mg/ animal In every 2^{nd} day for up to 2 weeks may give remarkable results because of their

antinflammatory used as an aid in the temporary treatment of COPD such as:

a- isoprenaline inhalation is a sympathomimetic drug which stimulate beta-1 (cardiac) and beta2 (smooth muscle) receptors causing cardiac stimulation and bronchial muscle relaxation (temporary relied for 1-2 hrs.)

b- Turbutaline inhalation is a sympathomimetic drug which exhibits action selectively in smooth muscle receptors (beta-2) causing bronchiodilation with no cardiac stimulation for 1-2 hours.

c- Clenbuterol HCL(long acting bronchodilator(is a beta-2 sympathomimetic has no untoward effect on circulatory system of exercising horses.

d- Atropine is a parasympatholytic drug given In at rate of 0.02 mg/kg B.w.

5- Antibiotics are used in treatment of COPD in horses, but there is only limited clinical evidence of their value. E.g. penicillin procaine 25.000 I.U/kg B.W. daily for 2 weeks (sulphamethazine has also been recommended).

PLEURISY (PLEURITIS)

Definition:

- Acute, subacute or chronic inflammation of the pleura.
- Acute pleurisy causes severe pain during respiratory movements, manifested clinically by shallow rapid respiration subacute pleurisy is accompanied empyemia causing collapse of the lung and respiratory embarrassment. Chronic pleurisy is usually manifested by the development of fibrous adhesions and interference with respiratory movement.

Etiology :

(A) primary cause:

- Traumatic penetration of chest wall.

(B) secondary causes:

1- Secondary to pneumonic lesion.

2- Rupture of a pulmonary abscess.

3- Streptococus equi infection (secondary to strangles)

4-pleuropneumonia in mature horses is due to anaerobic bacteria (Bacteroids spp.) and klebsiella pneumonae and streptococcus zooepidemicus.

5- Mycoplasma felis.

6- Equine infectious anemia (rare cause)

Clinical findings:

(A) Acute pleurisy:

1- Pain and anxiety manifested by rapid shallow respiration.

2- Respiratory movements are markedly abdominal.

3- Abducted elbows.

4- pleuritic friction sound in auscultation.

5- Deep digital palpation of intercostals space causes pain.

6- Elevated temp. and pulse rate.

7- Toxemia with anorexia and depression.

N.B:

- Acute pleuritis may causes colicy signs in horses

-Advanced pleurisy may causes subcutaneous oedema of the ventral body wall extending from the pectorals to the prepubic area (due to blockage of lymphatics drain the sternal lymph nodes.

(B) Subacute pleurisy :

1- Dullness on percussion over the fluid-filled area of the thorax and dull area has a level top line – a fluid line.

2- Dyspnea is evident, particularly during inspiration.

3- More severe toxemia and anorexia.

4- Elevated temperature, and pulse rate.

5- Painful, short and shallow cough (due to concurrent pneumonia)

(C) Chronic pleurisy :

1- Weight loss is one of the most common complains.

2- respiratory embarrassment because of the presence of adhesions (race horses do not regain complete efficiency).

3- rupture of adhesions during severe exertion may cause fatal hemorrhage,

Diagnosis :

(I) History ,

(II) Clinical signs

(III) Lab. diagnosis

1- Thoracocentesis to obtain a sample of pleural fluid.

2- The pleural fluid should examined for odour, color, viscosity, protein concentration, blood and bacteria.

3- The pleural fluid should be cultured for both aerobic and anaerobic bacteria and mycoplasma.

IV- radiographic examination :

Radiographic examination of chest may reveal the presence of fluid line and displacement of mediastinum and heart to the unaffected side and collapse of the lung.

V- Pleuroscopy :

Treatment:

- The prognosis of pleurisy in horses is unfavorable.

1- Control the infection by parenteral or oral administration of antibiotics or sulphonamides (detected by bact. Exam. Of pleuritic exudates)

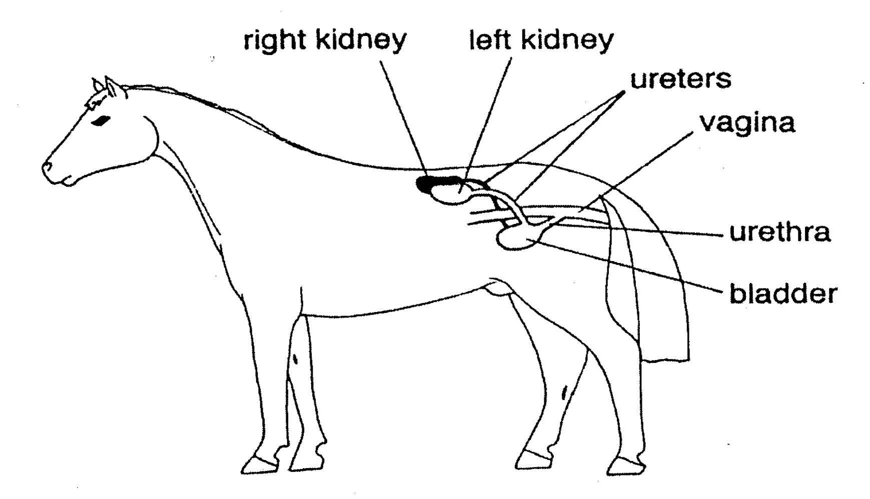
N.B:

Treatment of anaerobic bacterial pleuropneumonia in the horse may require the use of antimicrobial such as chloramphenicol or metronidazole.

2- Drainge or aspiration of the pleural fluid by special needle, but the use of diuretics is most useful and less dangerous.

3- Analgesics such as phenylbutazone in acute cases.

DISEASES OF URINARY SYSTEM



Glomerulonephritis

1- Glomerulonephritis is an inflammatory disease involving the kidney, affecting principally the glomeruli and may extending secondarily into the surrounding interstitial tissue and blood vessels..

2- It is a rare disease in animals (principally a disease of humans), occurs) in horses in association with:

a) Streptococcal equi infection.

b) Equine infectious anemia virus.

3- Glomerulonephritis usually resulting in chronic renal failure.

4- The most consistent signs of chronic renal failure in horses is "weight loss" additional signs are: anorexia, polyuria, polydipsia and ventral oedema.

5- The consistent lab. Diagnostic findings is a high blood urea nitrogen (BUN) and serum creatinine.

N.B:

Urine analysis findings vary with the nature and stage of the disease.

CYSTITIS

Definition :

Cystitis means inflammation of. m.m. of U.B. characterized by frequent, painful urination.

Etiology:

1- Introduction of infection into U.B. when trauma to bladder has occurred or when there is stagnation of urine e.g.

- Vesical calculus
- Contaminated catheterization.
- Paralysis of the bladder.
- Late pregnancy.

2- The infection causing cystitis usually mixed bacteria but predominantly E.coli.

N.B:

Sudax or sudan grass causes outbreak of cystitis in horses. It is causes possibly by a fungal toxin.

Clinical findings :

1- Painful sensation and desire to urination (due to urethritis which usually accompanies cystitis).

2- Frequent painful urination.

3- The horse remain in the posture adopted for urination for some minutes after the has ceased.

4- The volume of urine passed on each urination is usually small.

5-In very acute cases there may be moderate colicy pains and moderate febrile reaction.

N.B:

In chronic cystitis there is inflammatory thickening of U.B. wall which is palpable on rectal examination.

Diagnosis:

(I) History

(II) Clinical signs

(III) Lab. Diagnosis:

1- Microscopic exam. Of urine sediment reveals the presence of blood cells (erythrocytes), pus cells (leukocytes) and desquamated epith. Cells.

2- Bacteriological exam. (isolation and culture and sensitivity test) of urine confirm the diagnosis.

Treatment:

1- Irrigation of U.B. using mild antiseptic solution e.g. potassium permanganate 1 : 5000.

2- The use of drugs which causing change in pH or urine (have bacteriostatic action) e.g. Hexamine and mandelic acid.

3- Antibiotics, based on culture and sensitivity control infection for 7-14 days.

4- Free access to water to ensure a free flow urine.

Paralytic Bladder

Etiology :

Lesions in the lumbosacral part of the spinal and e.g.

- Neuritis of the cauda equina.
- Enzootic equine ataxia – cystitis (sudan grass or sorghum ataxia – cystitis which contain HCN).
- Fractures, ostemyelitis or neoplasia involving the lower, sacral or upper coccygeal vertebrae.

Clinical Signs :

1- in the early stages, the U.B remain fully and dribbling (urinary incontinence) occurs especially during movement, exercise or even coughing, which increase the intrabdominal pressure.

2- In the later stages, the U.B. will begin to empty involuntarily although evacuation is usually incomplete and some urine is retained causing cystitis .

3- Scalding of perineal area and rear limbs may be present in mares.

Treatment :

Prognosis of paralytic bladder is poor.

1- Essential regular catheterization (but avoid introduction of infection)

2- Adm. of antibiotics as a prophylaxis against the development of cystitis and may be a useful therapy for osteomyelitis.

3- Corticosteroid (0.5-1 mg/kg prednisone orally or 0.1 mg/kg dexamethzone injection I/m) every other day for 8-10 days if equine herpes virus-1 myeloencephalitis suspected.

4- Application of petroleum jelly to rear limbs to prevent urine scalding.

UROLITHIASIS (URINARY CALCULI)

Calculi may occur anywhere in the urinary tract in horses, but are found principally in the U.B. or urethra.

Etiology :

1- The pressure of mucous and epith. debris in equine urine leads not only to viscous and turbid appearing urine, but also may serve as a nuclei for calculi

2- Two types of calculi are commonly found in equines.

a) The first occurs in horses fed hay, is yellow – brown and crystalline, and composed principally of calcium carbonate.

b) The second from in horses being fed grain ration, this type is smooth white and composed of phosphates.

Clinical findings :

(a) Bladder calculi

- The initial calculi from in the renal pelvis and pass to U.B., where they increase in size by mineral deposition around the initiating nuclei of white or red blood cells, albumin, fibrin or epith cells.

- The horse may exhibit signs of abdominal discomfort when exercised, or may void urine accompanied by straining occasional blood in the urine.
- Rectal exam. Identifying the calculus in the bladder.

b) Urethral calculi

1- Restlessness, abdominal pain, and frequent urinary attempts.
2- On rectal exam, the U.B. found distended
3-The stone may be located anywhere in the urethra, but is more commonly found at the turn at the pelvic inlet.

Treatment:

Smooth – muscle relaxant may help the passage of stone, otherwise surgery is indicated.

RENAL FAILURE

Renal failure may be acute or chronic

1- Acute Renal Failure

Definition :

Acute renal failure is the inability of the kidney to perform the major functions of clearing nitrogenous waste products from blood and maintaining fluid and electrolyte haemeostasis. It is sudden and reversible. It is characterized by the clinical signs of azotemia (accumulation of nitrogenous waste products in blood) and biochemically by elevation of serum creatinine more than 1.0 mg/dl or urea 10 – 20 mg/dl. This usually occurs 2/3 or 3/4 of the nephrons are not functioning properly.

Incidence :

Uraemia or azotemia may be:

1 – Prerenal - decreased blood flow to the kidneys

2- Renal - various affections including glomeruli, ascending tubules, descending tubules, and collecting tubules.

3- Postrenal - impairment of urine flow after the kidney i.e. ureter, bladder and urethra.

Etiology:

1- Prerenal azotemia:

It may be usually due to

1- Hypovolemic reduction in cardiac output following circulatory failure.

2- Hypotension vascular thrombosis.

All these conditions may lead to severe ischaemia of the kidney followed by severe kidney damage, tubular dysfunction and may be eventually necrosis of kidney tissues.

II- Renal azotemia:

It is usually due to circulating potential renal causes due to

1-Nephrotoxic substances.

a- Endotoxic circulating substances: the endotoxins are usually due to systemic coagulopathy vascular damage or thrombosis such as myoglobin, which is one of the most contributing causes

b- Extrinsic nephrotic substances as

1-Medicaments

- Antibiotics and sulphonamides

aminoglycosides, sulphonamides, cephalosporin polymxin B, vancomycin, amphotricin B. Neomycin is most nephrotoxic while streptomycin is the least and kanamycin and gentamycin are intermediate

-Phenybutazone (prostaglandin synthetase inhibitors)

- Vitamins: - Vitamin D2, D3

-Synthetic Vit. K (Menadione sodiumbisulfate)

2- Heavy metals: as mercury, arsenic, selenium, canthridine (blister beetle), tetrachlorodibenzodioxin (Dioxin) and oxalate.

3- Bacterial infection of kidney:

It is difficult to be diagnosed until be fulminant.

The root of infection is either ascending or haematogenous. Chronic inflammatory condition in equine are usually caused by Corynebacterium equi, corynebacterium pneumonia, strept. abscess, equine infectious anaemia, leptospira infections, deposits of circulation antibody complex that cause glomerular damage.

III- Postrenal causes :

Include abnormalities in the lower urinary tracts as:

1- Rupture of the bladder that mainly occurs in neonatal foals and during parturition in mares.

2- Urethral and ureteral tears (laceration)

Affected equine shows anuria, Azotemia, dehydration, hyponatremia, hyperkalemia, and hyperchloremia. obstruction in one ureter is not manifested clinically due to compensation by the other kidney.

3- Calculi :

In horses, it is usually in the bladder and become lodges in the urethra of equine with those affections show dysuria, stranguria and occasional haematuria.

Clinical signs

These are non specific symptoms

1- Anorexia
2- Depression and weakness
3- Decrease performance
4- Mild to severe colic
5- Intermittent fever
6- Dehydration.
7- The urine may have abnormal flow or amount or constituents. These may be manifested as anuria or oliguria or change in color or consistency of urine:
    a. Normal urine outflow is 1-2ml/kg/hour.

b. Anuria, dysuria and stranguria suggest obstructive urinary problem.
c. Oliguria usually in case of haemodynamic causes.
d. Polyuria is the most common with aminoglycosides.

8- Increased water intake (polydipsia).
9- Oedema
10- Hypertension.
11- Kidney palpation is variable.

Diagnosis:

1- History
2- Symptoms
3- Ultrasonography
4- Renal biopsy.
5-Laboratory Diagnosis:

a- Urine analysis:

- Physical examination of the urine color is not specific for acute renal failure except reddish color indicates haematuria and myoglobinuria.
- On sedimentation analysis, there is high count of W.B.Cs if the causes infections.
- Bacteriological examination of urine for isolation, identification and drug sensitivity test.
- Another biochemical analysis of urine includes detection and estimation of protein, glucose, blood billirubin, ketone bodies and nitrates.
- Activity of gammaglutamyl transpeptidase (GGT) indicates destruction of renal tissues. A ratio of GGT to urine creatinine less than 2.5 is normal, but above this level indicates proximal tubular cell damage.

b- Whole blood examination.

c- Serum analysis:

- Serum sodium, potassium and chloride are within normal range in early stage.
- Hyponatremia, hypochloremia and hyperkalemia occur in insufficiency.

- Hypercalcemia and hyophoosphatemia had been found in bilateral nephroectomized ponies.
- Increased serum urea

Treatment :

I- Supportive treatment

1- Fresh clean water supply.

2- Feeding by stomach tube to minimize breakdown of endogenous protein.

Maintenance oral eleçtrolyte mixture for one day/450hg horse

Substance	Amount gm
Sodium chloride	10
Sodium bicarbonate	15
Potassium chloride	75
Dibasic potassium. Phosphate anhydrous	60
Calcuim chloride	45
Magnesium oxide	25

Intravenous feeding solution formulation

Item	Amount
5% Amino acid solution	1000ml
50% dextrose	500ml
Potassium	30mEq
Sodium bicarbonate	30mEq
Injectable multivitamins	

administration at a rate of 3L/day for 450kg horse

2- Fluid therapy: the amount of fluid therapy is detected by estimation of state of hydration using PCV or total plasma proteins. The amount of fluid therapy is 6-8 liters per os and can be repeated every 30 to 60 minutes. If no response, i.v. injection is required. If 450kg horse has 10% dehydration, the fluid required is 45 liters. This volume can be given rapidly at a rate of 10 liters/hour. If rehydration appears and the animal still anuric or oliguric, the volume of fluid must be restricted to 10 – 15 liters.

- The lung must be auscultated to avoid lung oedema.

IV and oral electrolyte supplements

Fluid i.v	Sodium mEq/L	Potassium mEq/I	Chloride mEq/I	Bicarbonate mEq/I	Calories
Saline	154	-	154	-	-
Lactate ringer's	130	4	109	28	9
5% dextrose	-	-	-	-	-
5% sodium Biacarbonate	600	-	-	600	-
Oral supplements					
Sodium chloride	284	-	284	-	-
Potassium chloride	-	217	217	-	-

II – Medical treatment :

-Bacteriostatic antibiotic with long halftime should be adjusted by increasing the dosing intervals while antibiotic with short half time should be given in lower dosage.

- Flunixine meglumine at 0.25mg/kg 3-4 times / day is effective against endotoxemia. (Fenadyne i.v. or.i.m)

- If the horse does not urinate, give 10% dextrose and 20% mannitol until the horse urinate for flushing the tubules from debris.

2- CHRONIC RENAL FAILURE

Definition:

Chronic progressive renal disease causes continued loss of nephron function or population. Renal disease differs from renal failure by clinical signs as in renal disease the signs are not obvious until 2/3 or more of nephron functions are lost.

Classification of renal failure according to clinical and pathological findings into:

1- Primary glomerular disease.

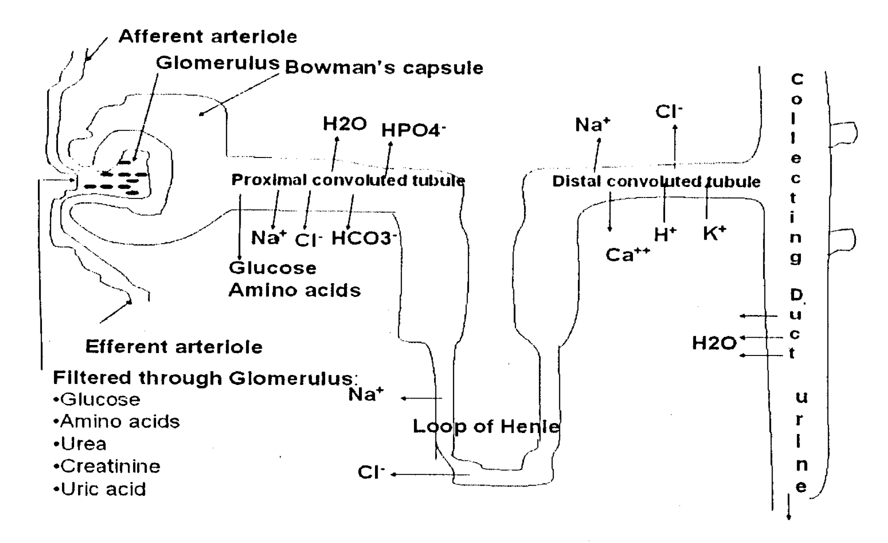
2- Primary tubulointerstitial disease.

Kidney function:

Two main functions of kidney:

1- Excretion of nitrogenous waste product e.g. urea and creatinine.
2- Regulation of water and electrolyte balance (homeostasis):
   a. The intial urinary filtrate in the glomeular vascular bed contains glucose, amino acids, water, electrolytes (Na, Cl, Ca, HCo_3 and HPO_4), Urea, creatinine, and uric acid.
   b. Reabsorption of glucose, amino acids, water, sodium, chloride, bicarbonate and phosphate occurs through proximal convoluted tubules.
   c. Reabsorption of sodium and chloride again occurs from the loop of Henle.
   d. Reabsorption of sodium, chloride and calcium; and excretion of potassium and hydrogen ions occurs in the distal convoluted tubules.

e. Reabsorption of water occurs in the collecting tubules under the effect of anti-diuretic hormone.



Causes:

I. Primary glumerular disease: Glomerulonephritis.

II. Tubulointerstitial disease occurs due to:

1-Acute tubular necrosis due to nephrotoxic substances as aminoglycosides, heavy metals and pigments, ischemia and hypovolemia.

2-Pyelonephritis, hydronephrosis and interstitial nephritis.

3- Abnormal high mineral excretion may cause nephrolithiasis.

Pathophysiology:

Renal diseases
Primary glomerular
Primary tubulointerstitial

↓ Total destruction and absence of function of some nephrons ↓ Partial damage and decrease of function of nephron

Decrease in the number and/or function of nephron

↓

Decrease glomerular filtration rate (GFR)

↓

1- Decreased filtration of protein byproduct (urea, creatinine and guanine).
2- Failure to catabolize certain hormones e.g. PTH

↓

Accumulation of byproduct in plasma to toxic level

↓

Destruction of renal tubules

↓

1-Inability to reabsorb Na^+, Cl^-, $HPO4^-$.
2- Inability to excrete H^+ and K^+
3-Inability to reabsorb water
4- inability to reabsorb Ca^{++}

↓

1-Hyponatraemia, hypochloremia, hypophsphatemia
2- Acidosis and hyperkalemia (exchange of H^+ with K^+ in muscles aggravates the hyperkalemia).
3- Ployuria, polydipsia and dehydration
4- Initial hypocalcaemia ⟶ Stimulate PTH

↓

Stimulates osteoclast activity

↓

Increase Ca levels

Uremia leads to:

1-Moderate anemia due to toxic effect on RBCs and due to decreased erythropoietin

2-Its effect on gastrointestinal tract

a) The increased serum urea may excreted in salivary secretion leading to urinephrous odour or breath

b) Abnormal oral flora and excessive dental. tarter. Also focal ulceration of oral and intestinal mucosa.

3-Hyperlipaemia: because azotemia inhibits peripheral removal of triglycerides from blood. Also lipolysis may occur with anorexia.

Clinical signs

1 -Weight loss 2-Inappetance

3-Dysuria 4-Fever in urinary tract infection

5-Symptoms of uremia.

6-Rectal examination reveals smaller kidney and non obstructive tubulointerstitial disease.

7-Ureteral obstruction and pyelonephritis produce enlargement of kidney and ureters.

Diagnosis :

1-Case history 2-Symptoms

3-Ultrasonography 4-Renal biopsy

5-Lab. Diagnosis. It is variable

a-Azotemia: moderate increased blood urea nitrogen and creatinine while high increase may be due to pre or post-renal factors

b-hyponatremia, hypochloremia and hypophosphatemia.

3-Hypercalcemia.

4-Leucocytosis.

5-Anaemia.

6-PCV is 20-30%

7-In pyelonephritis there is proteinuria and WBCs count increase

8- Urea to creatinine ratio more than 15:1

Treatment :

1-water supply must be sufficient in the hope of maintaining normovolemia.

2-Mineral block should be applied if there is no oedema or hypertension

3 -If plasma bicarbonate less than 18 Meq/L. up to 225mg/day of sodium bicarbonate is added to water or food.

4- If plasma bicarbonate is more than 24 meq/L and chloride concentration is decreased. Additional sodium chloride should be added to the diet

5- If hyperlipermia is marked. Heparine 40-100 iu/kg s/c twice daily can be used

6- Supportive therapy in polyuric patients passing more than 18ml urine /kg B.W/day,

7- No more protein in diet for uremic patients.

DISEASES OF THE CARDIOVASCULAR SYSTEM

DISEASES OF CIRCULATORY SYSTEM

-The two functional units of C.V.S.are:

1- Heart

2- Blood vessels (arteries veins and capillaries)

-The heart and blood vessels giving rise to two forms of circulatory failure:

1-Heart failure.

2-Peripheral failure

Manifestations of circulatory failure:

(I) Acute heart failure

(II) Congestive heart failure.

(III) Peripheral circulatory failure

I-ACUTE HEART FAILURE

(Cardiac syncope)

Definition:

It is an acute cardiac disorder characterized clinically by:

-Sudden loss of consciousness.

-Falling with or without convulsions.

-Severe pallor mucosae (m.m).

-Either death or complete recovery from the episode.

Etiology:

A- Severe defect in filling (disorder of filling)

B-Failure of the heart as a pump either due to severe tachycardia or bradycardia.

C-Sudden increase in work-load.

(A) Disorder of filling

Due to pericardial tamponade (compression of heart for instance by haemopericardium)

(B) Failure of heart as a pump

occurs due to either tachycardia or Bradycardia

a-Tachycardia:

I-Myocarditis e.g. encephalomyocarditis, v.

2-Nutritional deficiency myopathy e,g. Copper and selenium deficiency

3-Electrocution strike.

B-Bradycardia

1-Dosed by rapid I/V calcium preparation

2- Sudden increase in work-load

Occur due to:

1-Acute anaphylaxis shock

2- Rupture of aortic valve.

N.B:

Acute heart failure due to occlusion of the coronary vessel in man as recorded rarely in

Pathogenesis

1-In disorder of filling due to pericardial tamponade and in tachycardia due to shorting of diastolic period, the filling of the ventricles is impossible, therefore cardiac output grossly reduced,

2-In severe bradycardia, the cardiac output also grossly reduced.

3-In sudden increase in workload cardiac fibrillation occur therefore no coordinated contraction thus no blood ejected from the heart.

4-In all of these circumstances there is a fall in minute volume of the heart which lead to severe degree of tissue anoxia.

-The brain is the most sensitive organ first affected; therefore, clinical signs are principally nervous type

-Pallor occur due to reduction in arterial blood flow

Clinical Findings:

1-Sudden loss of consciousness due to cerebral anoxia.

2-Staggering and falling with or without clonic convulsions .

3-Marked pallor mucosae (m.m)

4-Death usually follow within seconds or minutes and usually accompanied by deep, asphyxial gasps.

5-If there is time for exam., there is. Absence of pulse and there is either tachycardia brady cardia or absence of heart sounds.

N.B:

In less acute cases the course may be as long as 12-24 hours, and dyspnea and pulmonary oedema are prominent signs.

Diagnosis:

(I) History

(II) clinical signs.

N.B:

Insufficient time is available to conduct lab. Diagnosis.

Treatment is not usually possible because of the short course of the disease, but the following may be useful.

1- Direct cardiac massage or electrical stimulation but these techniques are restricted to the more sophisticated surgical units.

2- Intracardiac injection of very small doses of adrenaline and as much harm as good.

II-Congestive Heart failure

Definition:

It is a cardiac disorder characterized by "congestion of the venous circuit" accompanied by"

1-Enlargement of the heart and increase in heart rate.

2-Dilation of veins and odema of lungs or periphery.

Etiology:

Diseases of the endocardium, myocardium and pericardium

1-Interfere with flow of blood into heart or away from the heart.

2-Diseases which impede the heart action

(A) Endocardial diseases

- Comprise diseases of the valves e.g.

1-Aortic and pulmonary valve stenosis.

2-Aortic and mitral valve insufficiency

(B) Myocardial diseases

1 -Myocardial asthenia (weakness in myocardium)

2-Myocardial arrhythimia (irregulatory in rhythmic control)

e.g:

1 -Myocarditis in african horse sickness in equine

2-Myocordral dystrophy in copper, selenium and vit. E deficiency.

3-Myocardial neoplasms.

(C) pericardial disease

Comprise, hydropericardium, cardiac tamponade and pericarditis.

Clinical signs.

-Generally in very early stages of C.H.F., there is respiratory distress on light exertion and time required for return to normal respiration and pulse rates is prolonged.

- C.H.F. may be referable to failure of left side of the heart. or right side of the heart.

A) Left sided C.H.F. (Signs of lung oedema)

Characterized by:

1- Increase rate and depth of respiration rest.

2-Moist cough, and mucoid bilateral nasal discharge plus frothing from mouth.

3-Moist rales at base of the lungs.

4-Increased dullness on percussion of ventral border of lungs.

Diseases of the endocardium, myocardium and pericardium which

5- Terminally. Severe dyspena and cyanosis.

6- Tachycardia and there may be a murmur (referable to left atrioventricular (mitral) or arotic semilunar valves.

7- Epistaxis in severe csases.

8- Corneal opacity (glucoma).

(B) Right sided C.H.F (Body oedema)

1- Oedema (anasarca, ascitis, hydrothorax and hydropericardium.

2- Anasarca charactestically limited to the ventral surface of the body, the neck and the jaw.

3- Tachycardia and dilation of superficial veins (particulary J.V.)

4- Faces normal at first, but in the late satge profuse diarrhea may be present.

5-Oliguria and albuminuria.

N.B:

Live severely enlarged and protruding beyond the right costal arch in severe right sided C.H.F.

Diagnosis:

I- History.

II- Clinical signs (observation, palp., percussion, Auscaltation).

III- Venipuncture test.

Increased pressure of blood from needle on vene- puncture much greater than normal due to increase venous pressure.

IV- Radiography.

V- ECG.

VI- Echocardiography.

Treatment:

Non-specific treatment is applicable in most cases irrespective of the cause. It consists of:

1-restricition of activity (to reduce the demand on cardiac output).

2-Diuretic medication (to overcome the load of oedema) orally or I.M.

3- Digitalis glycosides (to improve myocardial contractility).

4-Venesection can be used as an emergency treatment in acute pulmonary oedema (4-8 ml of blood / kg B.W may be withdrawn).

5-Paracentesis for drainage of serous cavities.

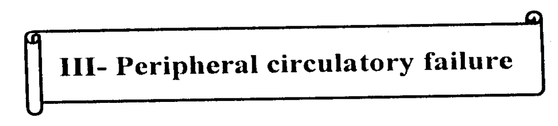
Digitalization in horses:

Digoxin given I/V according to the following schedule:

1- Initial digitalizing dose 1.6 - 2.2 mg/100 kg I/V followed by

2- Second dose (0.5 digitizing dose) every 4 hours followed, by

3-Third dose (0.5 digitizing dose) every 24 hours.



Defintion:

-It is a circulatory disorder characterized by reduction in cardiac output due to failure in the " Venous return ".

Etiology:

Failure in the venous return may be:

1- Vasogenic failure 2- Haematogenic failure

(A) Vasogenic failure

1-Occur when there's peripheral "Vasodilation" and pooling of blood in B. vessles.

2-This is principally occur in "Shock when the blood collected in the dilated splanchic B. vessles.

(B) Haematogenic failure (Hypovolemic failure)

1- occur when there is reduction in the circulating blood volume

2- This occur principally in:

a) Hemorrhage b) Dehydration

Clinical findings:

Failure of venous return, lead to reduction in cardiac output which in turn lead to decrease blood flow to tissues resulting in tissue hypoxia or anoxia which in turn responsible for the following clinical manifestations:

1- Anorexia, which may accompanied by thirst.

2- Shallow rapid respiration.

3- Tachycardia accompanied by weak intensity of heart sounds, as well as, abnormalities of pulse amplitude.

4-Low arterial blood pressure (measured either directly by arterial puncture or by indirect methods using sphigmography).

5-Nervous signs include depression, weakness and restlessness, as well as, coma in the terminal stages.

N.B:

Clonic convulsions may occur but they are not a prominent dart of the syndrome.

6- Cold skin and extremities.

7- Pale m.m with prolonged capillary filling time.

8- Subnormal temperature.

Diagnosis:

I-History

II- Clinical signs

III- Lab. Diagnosis.

1- PCV, Hb, and total erythrocytic count, usually below normal due to stroke, haemorrhagic anemia and dehydration.

2- Eosinopenia, lymphopolymorphocytopenia and thrombocytopenia.

3- Hyperkalaemia.

4- Commonly with P.C.F there's disturbance in the acid base status with metabolic acidosis and lactic acidosis.

Treatment:

-Regardless of the cause, treatment is to restore the circulating blood volume to normal to avoid tissue anoxia.

(a) In Vasogenic Failure

Plasma transfusion is required to overcome shock.

(b) Haematogenic Failure

1- Give whole blood transfusion to overcome hemorrhage.

2- Give isotonic fluid replacement overcome dehydration.

N.B:

1- In vasogenic failure, a large dose of coticosteriods (2-3 mg / kg I/V) may be useful as an anticardiogenic shock.

2-In both vasogenic failure and haematogenic failure, a specific bicarbonate (Na bicarbonate 1% I/V) should included to overcome the disturbance in acid-base status i.e marked blood acidosis and lactic acidosis.

Diseases of C.V.S

A- Diseases of heart

1- Pericarditis

2- Myocardial asthenia

3- Rupture of the heart

4- Endocarditis

5- Valvular dieases

6- Cor pulmonale

7- Congenital defects

B- Diseases of B.V

1- Arterial thrombosis

2- Venous thrombosis

3- Hemorrhagic diseases

4- Haemangioma

I- Pericarditis

Definition:

Pericarditis means inflammation of the pericardial sac which usually resulting in "congestive heart failure" and toxemia.

Etiology:

1- Localization of blood-born infection in the pericardium.

2- Extension of infection directly from myocarditis or pleurisy diseases encountered in horses and causing pericarditis tuberculosis, streptococcus spp. And idiopathic fibrinous pericarditis.

Clinical findings:

(a) The early hyperaemic stage manifested by:

1- Avoidance of movement, abduction of elbows, arching of the back and shallow abdominal respiration.

2- Pain on firm palpation or percussion over the cardiac area of the chest wall.

3- Pericardial friction rib (sound) on auscultation of the cardiac area (due to hypermia of the pencardial sac and deposition of fibrinous exudate

4- Temp. may be slightly elevated in this early satge.

(b) The 2nd effusion (exudation) stage manifested by:

1- Increase in the area of cardiac dullness on percussion over the cardiac area (due to exudation pericardial sac)

2- If gas (form gas forming M.O) is present in the pericardial sac a splashing sounds present on each cardiac cycle.

3- Signs of right sided congestive heart failure (edema, tachycardia, and engorgement of superficial veins particularly j.v) become evident because

the accumulated fluid compresses the atria and right ventricle, prevent their complete filling.

1- Signs of severe toxaemia (antigenic) which depend on type of bacteria present.

2- Animal usually die of congestive heart failure of from toxaemia in 1-3 weeks.

(c) The 3rd chronic stage manifested by:

Animals that survive pass through a long period of chronic ill-health during which:

1- The heart sound become less muffled and fluid sounds disappear or persist in restricted area (because fluid resorbed in recovery stage).

2- Signs of congestive heart failure diminishes slowly.

3- Signs of toxaemia subsided relatively quickly.

4-Additional signs of myocarditis, particularly myocardial arrhythmia may appear.

5- Complete recovery is not common.

Diagnosis:

I- History

II- Clinical signs

III- Lab. Diagnosis.

Blood picture revealed marked leukocytosis and shift to left.

IV- Pericardial puncture:

Pericardial puncture in the effusion stage to aspirate sample of pericardial fluid and submitted for bact.Exam.

Treatment:

1- If possible, antibacterial treatment for the specific causes of infection or give broad-spectrum antibiotics or S.M. as a non specific treatment.

2- Repeated paracentesis to relive the fluid pressure in the pericardial sac, but this give only a temporary relief because the fluid returning quickly.

3- Diuretics is recommended, but digitalization is usually not very effective and is dangerous if infection is still present.

4-Surgiacl treatment, to provide continuous drainage of the sac, but not usually successful.

RUPTURE OF THE HEART

Etiology:

1- Spontaneously in race horses

2- Rupture of the base of the aorta just above the aortic valve. This because the wall of aorta may have been weakened by verminous arteritis associated with migrating strongylus in horses.

3- Rupture of the pulmonary atery also recorded, due to pulmonary hypertension.

Pathogenesis and clinical signs:

1- Rupture of the heart, aorta or pulmonary A . resulted immediately in filling of the pericardial sac with blood (cardiac tamponade).

2- cardiac tamponade result in sudden death due to acute H.F.

COR PULMONALE

Definition:

It is a syndrome of right heart failure resulting from an increase in right heart work load secondary to increased pulmonary hypertension.

Etiology:

Pulmonary hypertension which resulted from:

1- Chronic interstitial pneumonia and chronic alveolar emphysema due to partial destruction of the pulmonary vascular bed.

2- Chronic obstructive pneumonia due to airway obstruction and accumulation of fluid in distal airways, induce pulm. Hypertension by combination of hypoxia and reduction of the pulmonary vascular bed.

VENOUS THROMBOSIS

Definition:

Venous thrombosis mean development of thrombin the veins and may result in

1- Local obstruction to venous draining. Or

2- Liberation of emboli which may lodge in brain lung, liver or other organs.

Etiology:

Phlebitis is the common origin of thrombi which may be caused by:

1- Infection of the umblical vein in the porn foals.

2- Extension of infection from surrounding diseased tissue e.g strangles in horses, and commonly causing thrombosis of J.V and caudal V.C.

1- Localization of blood born infection e.g bacteremia and septicemia.

2- Injection of irritant drugs into major veins e.g phenylbutazone.

Clinical signs:

1- Engorgement of the vein.

2- Pain on palpation of area drained by the vein.

3- Local oedema.

4- In unsupported tissue rupture of the vein may occur lead of fatal internal or external hemorrhage.

N.B:

Oedema of perineum, udder and ventral abdominal wall may occur due to pressure (not phlebitis and thrombosis) of foetus during late pregnancy.

Treatment:

1- Parenteral antibacterial drugs to overcome phlebitis.

2- Hot fomentation to external veins to overcome obstruction and subsided swelling.

EQUINE METABOLIC DISEASE

(1) AZOTURIA

Paralytic myoglobinuria, Lumbago, Monday morning disease, Tying-up syndrome, Exertional rhabdomyolysis





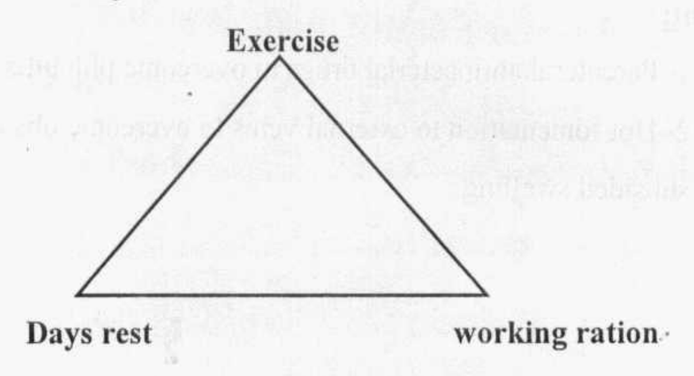
Definition:

It is multifactorial myopathy affecting mainly draft horses and less frequently the race horses. The disease is a metabolic muscular disorder of horses characterized clinically by stiffness in gait, lameness and swelling and hardening of massive muscles and biochemically by the presence of myoglobin pigments in urine (myoglobinuria).

Incidence, occurrence and predisposing factor:

1- The disease affects mostly draft horses (Draught) but race horses are sporadically affected.

2- During exercise after a period of at least 2 days of complete rest on a ful working ration.



Etiology and pathogenesis :

1-Carbohydrate Overloading

- The classical presentation is feeding the draught horse on full working ration during resting at weekend and when the horse returns to work several days later it suffers an attack of the disease.
- The muscle glycogen accumulates during the rest period and when used during exercise it produces excessive lactic acid in a rate higher than removal by blood vessels leading to lactic acid accumulation.
- This causes local tissue damage (myopathy) and constriction of the blood vessels, resulting in decreased blood flow to the tissues and further reduction in lactic acid removal.
- Accumulation of sarcolactic acid in muscles produces swelling and hardening of muscle (hard board-like).

2-Local Hypoxia

- Certain types of muscle fibers are larger, have greater glycogen stores and fewer surrounding blood vessels than others.
- Local hypoxia (lack of oxygen supplied by the blood) may increase the lactic acid production in these fibers.
- However equine rhabdomyelosis normally occurs at the start of exercise, when these fibres would not yet be working and the condition is not usually seen in horses with other conditions causing impaired circulation

3-Thiamine Deficiency

- Thiamine (one of the Vitamin B complex) involved in the metabolism of waste products from muscle activity.
- A deficiency, therefore, could lead to a build up of these waste products and hence, lactic acidosis.

4-Vitamin E and Selenium Deficiency

- This theory is based on reports of success at preventing further episodes following supplementation.

5-Hormonal Disturbances

- Reproductive hormones, thyroid hormones and cortisol have all been implicated in equine rhabdomyelosis.

6-Electrolyte Imbalances

- Chronic sodium and/or potassium deficiencies may be involved in chronic equine rhabdomyelosis.

Pathogenesis

1- During exercise the large store of glycogen formed during the period of rest in the muscles metabolized to sarcolactic acid.

2- Accumulation of lactic acid leads to:

a- Degeneration of the muscles and liberation of myoglobin (muscle haemoglobin)

b- Swelling of muscle because lactic acid is hydrophilic.

Clinical signs:

Signs develop 15 -60 min after beginning of exercise. Clinical signs vary according to the amount of exercise and extend of muscle damage:

A- In very mild cases that receive little amount of exercise, only poor performance is observed.

B- In mild cases, stiffness in gait and shuffling are observed.

C- In severe cases, which receive excessive exercise:

1- Profuse sweating, stiffness in gait and reluctance to move. Then the horse ,assumes a dog-sitting position followed by lateral recumbency, laying down and repeated attempts to rise.

2- Accelerated weak pulse, rapid respiration and temp. may rise up to 40.5°C

3- Hard board – like muscles particularly of hind legs (gluteal and quadriceps femoris)

4- The horse voided dark – red brown urine.

N.B:

Prognosis is good if animal remains standing but death may occur in recumbent horse due to decubital septicemia (or bed) or myoglobinuric nephrosis and uraemia.

Diagnosis :

I- History

II- Clinical signs

III- laboratory diagnosis

(A) Urine analysis:

For detection of myoglobin pigments in urine and this only spectrophotometrically.

(B) Blood :

- For determination of the muscle specific enzymes CPK and SGOT. Both enzymes are usually elevated but the 1st enzyme is more specific and diagnostic than the other.

(c) Histopathology:

Hyaline degeneration of the heavy muscles(Zenker's necrosis) in histopathological examination of muscle Biopsy.

Treatment

I- Hygienic treatment

1- Avoid further exercise.

II- Supportive treatment

1- Massage of limbs by hot application.

2-One liter of 1% Na bicarbonate IV may be useful against blood acidosis and alkalization of urine.

3- I/V injection of large quantities of fluids and electrolytes to maintain high rate of urine flow to avoid renal tubule blockage and subsequent uraemia.

III- Medicated treatment

1- I/M chloral hydrate narcosis particularly if horse attempt to rise to avoid self-injury.

2- I/M injection of 0.5gm thiamine HCL daily to increase the tolerance of blood to lactic acid by increasing lactic acid metabolism.

3-Non-steroidal anti-inflammatory drugs (NSAIDs) such as flunixin and phenylbutazone may be used to control the pain.

4- Some drugs, such as Acepromazine (ACP) can be used to increase blood flow and alleviate muscle spasm.

5-Antihistaminics

6- administration of Vit. E and selenium may be useful.

N.B:

- Trypan blue injection for conversion of myoglobin to oxyhaemoglobin may be useful for normal respiration.

Control (prevention)

1 -Reduce the working ration (particularly grains) to 0. 5, the amount during the period of rest.

2-Exercise should start light then gradually increased.

II- LACTATION TETANY OF MARES

Transit tetany of mares, Eclampsia of mares

Definition:

It is a metabolic neuretic disease of lactating mares characterized clinically by stiffness in gait and tatany and biochemically by hypocalcaema.

Incidence, occurrence and predisposing factors:

1-Most cases occur in lactating mares, either at about the 10th day after foaling or 1-2 days after weaning.

2-Mares have a heavy flow of milk.

3-Pregnant mares subjected to hard physical work or exercise.

4-Pregnant mares during or after prolonged transport.

Etiology:

1- The basic constant biochemical finding is low serum calcium level (hypocalcemia) in which serum calcium level ranges between 4-8 mg%

2- Hypo- or Hypermagnesemia have been observed in some cases.

Clinical signs:

Clinical signs are related to the degree of hypocalcaemia, because:

1- When serum calcium level is higher than 8 mg%, the only clinical sign is increased excitability

2- At levels of 4-8 mg %, there are tetanic spasms.

3- At levels less than 4 mg%, there are recumbency and stupor (state of unconsciousness).

The clinical signs progress as follow

1-Profuse sweating

2-Muscular fibrillation particularly of the masseter and shoulder region

3-Trismus (spasmodic contraction of M. of mastication) but no prolapse of 3rd eyelid

4-Normal pulse in early stages, but later becomes rapid and irregular.

5-Rapid, violent respiration accompanied with wide dilation of nostrils.

6-Normal temp. or slightly elevated.

7-Dysphagia (unable to swallow)

8- OLiguria or even anuria and constipation.

9- Difficulty in moving, stiffness in gait and incoordination.

10-Within 24 hours, the animal goes down then tetanic convulsions develop and death may occur about 48hrs. after onset of illness due to respiratory failure.

N.B:

The disease may be confused with other 2 common equine diseases: tetanus and laminitis.



Fig.1. A horse with laminitis.

Diagnosis :

I-History

II- Clinical signs.

III-laboratory diagnosis:

Estimation serum calcium level usually between 4-8 mg% (normal around 10 mg%)

IV-Therapeutic diagnosis

Response to treatment with calcium preparations

Treatment:

I/V injection of 400-800m C.B.G 25% causes rapid, complete recovery. If no response, repeated after 12 and 24 hours.

N.B:

One of the earliest signs of recovery is the voiding of large volume of urine.

Prevention:

Single IV or S/C. 10 millions I.U. crystalline vit. D immediately after foaling and repeated at weaning time for lactating mares.

(11) Hyperlipemia of ponies

Definition:

It is a metabolic disease of ponies occurring mainly in late pregnancy or early lactation and clinical signs related to the widespread vascular thrombosis and hepatic and renal failure and biochemically the disease characterized by milk-like serum and hyperlipidemia..

Incidence, Occurrence and Predisposing Factors

1-The disease occurs principally in late pregnant ponies or ponies in early lactation (4-8 years of age after parturition).

2-Mares and stallions may be affected in association with other underlying diseases e.g. parasitism or sand colic.

Etiology:

1 -Basic biochemical finding is severe hyperlipidemia.

2-The factors initiating hyperlipidemia is obscure.

a)It may be the response of some ponies to fasting

b) It may be a nutritional stress during a period of high nutrient requirement (late pregnancy or early lactation.)

Pathogenesis:

1-It appear to be a disturbance in fat metabolism occuring.

a) Spontaneously as a result of nutritional deviation

b) Secondarily to some underlying disease.

2- Most cases of hyperlipemia are associated with reduced food intake. During fasting, adipose tissue triglycerides are broken down by a **hormone sensitive lipase** to glycerol and free fatty acids. A significant portion of the fatty acids are taken up by the liver where they may be completely oxidized via the TCA cycle, used for ketone production, or reesterified to triglycerides. Triglycerides either accumulate in the liver or are released into the plasma as very low density lipoproteins (VLDL). In equine; triglyceride

production is emphasized over ketone formation, therefore, lipemia rather than ketosis dominates the response to prolonged fasting.

3- Lipoprotein lipase, also a hormone sensitive enzyme, is present in the capillary vessels of adipose, muscle, heart, and other tissues. Triglycerides in plasma VLDL are hydrolyzed to fatty acids and glycerol which are transported into peripheral tissues. Lipoprotein lipase activity in hyperlipemic ponies has been found to have twice the activity of nonlipemic ponies, so the excess serum triglyceride concentration in hyperlipemia appears to be the result of increased hepatic production rather than impaired function of lipoprotein lipase.

4- A prolonged increase of serum triglyceride concentration is associated with lipid accumulation in the liver, kidney, myocardium, and skeletal muscles, and this impairs the function of these organs.

5- The clinical syndrome is a reflection of the severe hyperlipidemia and is associated with wide spread extensive vascular thrombosis.

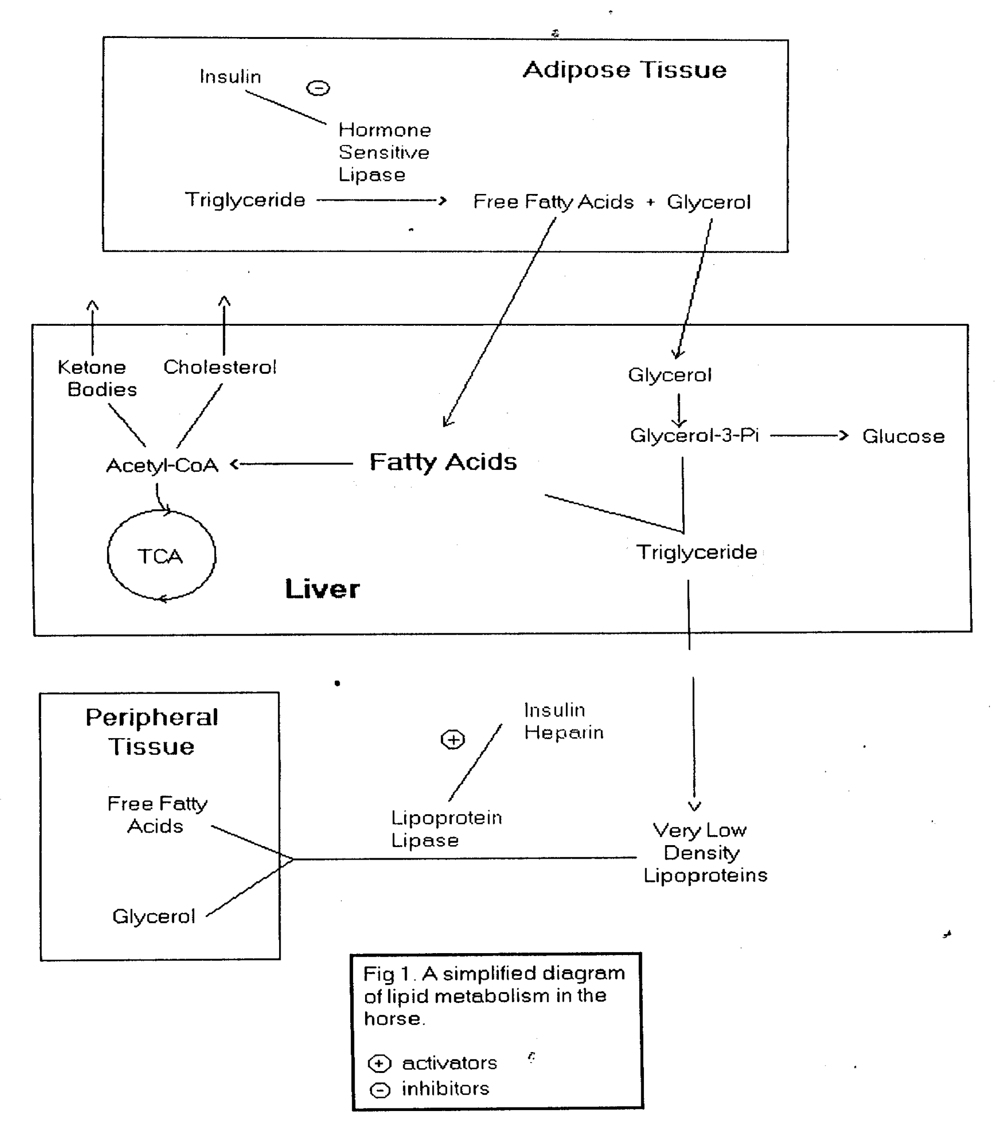


Fig 1. A simplified diagram of lipid metabolism in the horse.

⊕ activators
⊖ inhibitors

Clinical findings:

1 -The clinical course of the disease between 3 and 22 days (but is generally 6-8 days)

2- The initial signs are depressioin,, inappetence and weight loss.

3-Neuromuscular irritability with fine twitching of muscles of neck, trunk and limb (muscle fasciculation), sometimes there is terminal mania.

4-Ventral oedema in 30% of cases.

5-Depression progresses to somnolence and hepatic coma (but jaundice is not a feature)

6-Fetid diarrhea and metabolic acidosis

7-Temp.is normal or moderately elevated, pulse and resp. rates are increased.

Diagnosis :

I-History.

II-Clinical signs.

III-laboratory diagnosis:

-Serum or plasma show a milk- like opalescence due to hyperlipidemia.

1-Estimation of serum total lipids 4-8 gm%

2-Estimation of serum triglycerides (part of total lipids) is very high (30 times normal level)

3-Abnormal hepatic and renal functions.

4-Marked metabolic acidosis in terminal stages

5-Leukocytosis with neutrophilia.

6- PCV and Hb are elevated (except in cases with parasitism)

7-Hypophosphatemia in some cases.

N.B:

-Blood glucose is variable and abnormal glucose tolerance may be present.

IV Necropsy findings

- Extensive fatty changes is present in most internal organs.

Treatment:

Specific treatment of hyperlipemia is unrewarding and the case fatality is high.

1-Correction of the possible initiating factors e.g. parasitism and nutritional stress may induce recovery

2-A regimen consisting of administration of 30 I.U. insulin parentrally with 100gm glucose orally and 15 I.U insulin, parentrally with 100 gm glucose orally on alternative days has been reported to decrease the fatality rate. Correction of the metabolic acidosis

Control:

1-parasites and disease control program to ponies in late pregnancy and early lactation.

2- Give the nutritional requirement to ponies during late pregnancy and early, lactation.

3-Avoid transportation of ponies during late pregnancy and early lactation.

DISEASES OF NERVOUS SYSTEM

Modes of nervous dysfunction:

1-Excitation or irritation signs

increased activity of the organ occurs because of an increase in the number of nerve impulses received either because of excitation of neurons or by facilitation of the passage of stimuli. The excitability of nerve cell can be increased by many factors including stimulant drugs. Inflammation and mild degrees of those influence which in a more severe form may cause depression of excitability as anoxia and hypoglycemia. The major manifestation of irritation of nervous tissue are convulsion and muscle tremor in locomotor system and hyperaesthesia and paraesthesia in the sensory system

2-Release signs

exaggeration of normal nervous system activity occurs when lower nervous center are released from the inhibitory effects of higher centers cerebellar ataxia is a classical example. In the absence of cerebellar control combined limb movements are exaggerated in all mechanisms of actions including rate, range force and direction

3-Paralysis due to tissue destruction.



Muscles and tendons of near fore leg of horse from elbow downward.

Different degrees of depression of activity can occur with of nerve cells. The terminal stage being complete paralysis when nervous tissue is destroyed such depression of activity may result from failure of supply of oxygen and other essential nutrients either directly from their general absence or indirectly because of failure of local circulation. Infection of the nerve cell itself may cause initial excitation. Then depression of function and finally complete paralysis when the nerve cell dies signs of paralysis are constant and manifested by muscular paresis when the motor system is affected and by hypoaesthesia or anaesthesia when the sensory system is involved.

Nervous shock

An acute lesion of the nervous system causes damage to the nerve of the lesion in addition temporary cessation of function in parts of the nervous system not directly affected the loss of function in these areas is temporary and usually persists for only a few hours. Stunning is the obvious example.

Encephalomyelitis

Definition:

It is an inflammation of the brain and spinal cord. It is highly fatal disease characterized by initial signs of irritation followed by signs of loss of nervous function.

The loss of nervous function of motor nervous system in the form, paralysis while the loss of nervous function of sensory nervous system includes somnolence, lassitude, syncope and coma.

The nervous onset of unconsciousness is sudden in syncope and more gradual in coma. Hyperaesthesia may result if the dorsal nerve nuclei are involved.

Etiology:

1-Eastern and western equine encephalomyelitis they are viral diseases transmitted by mosquitoes

2 -Equine protozoa, protozoal myeloencephalitis : it is caused by coccidian parasite similar to sarcocyst.

Clinical signs

1- In case of viral infection, there are fever, anorexia, depression, and tachycardia.

2- Change in the behaviour of the animal. Head pressing teeth grinding circling and blindness.

3- Intense pruritis and hyperexcitability.

4-Cranial nerve dysfunction may produce nystagmus, facial paralysis and dysphagia.

5- Progressive ataxia,.weakness and inability to stand

Diagnosis:

1 -History and clinical signs.

2-Postmortem and histopathological examination 3-3- laboratory examination

a- For viral encephalomyelitis

1 -Virus isolation from fresh refrigerated or deep-frozen portion of the brain

2-Serological examination includinghaemagglutination inhibition test (HI) and complement fixation test (C.F.)

3-Examination of blood

-Transient leucopenia during the initial viremic stage.

-Increase PCV and hyperproteinemia if the horse is dehydrated.

-Elevation of serum bilirubin if the horse was anorectic for few days

4-Examination of cerebrospinal fluid reveals increase in total protein a-rid mononuclear leucocytes.

b-For protzoal myelocencephalitis, detection of the coccidian parasite similar to sarcocyst.

Treatment:

1-Adequate food and water intake should be ensured. nasogastric tube feeding can be employed to maintain hydration and provide energy. If the horse can not eat nor drink, Iv fluids will also be used

2-A well- bedded area should be provided to minimize the complication of recumbency

3-To avoid fatal cerebral oedema, we can use

-IV dimethylsulfoxide 1gm/kg as 2-40% solution in 5% dextrose once a day for 3 days, Dimethylsulfoxide has both diuretic and anti-inflammatory effect.

-Slow IV administration of mannitol 20% as 0.25 to 0.5gm%kg

-Furosemide also can be administered.

4-Anti-inflammatory drugs as dexamethazone can be used as 0.1 – 0.2mg/kg four times daily.

5-in highly feverish cases. We can use antipyretics as dipyrone 11:22 mg/kg BWt.iv and phenylbutazone, (Phenobarbital) diazepam and chloral hydrate.

6- Broad spectrum antibiotic in case of viral encephalomyelitis we can use pyrimethamine and sulphonamides as they can pass blood brain barrier

-Oral pyrimethamine (0.1 --0.2 mg/kg once daily)

- Trimethoprim and suaphadiazine have a significant effect.

- Oral trimethoprim-sulphadiazine (15 mg/kg twice daily)

Narcolepsy:

Definition:

Narcolespy is an incurable non progressive central nervous system disorder characterized by abnormal sleep tendency including excessive daytime sleepiness and pathological manifestations of rapid eye movement sleep. In narcolepsy muscle relaxation intrudes abruptly into wakefulness. This muscle

weakness produces a collapse or cataplexy which is the most obvious objective sign of narcolepsy.

Clinical signs:

1- The disease is seen in horses of all ages.
2- The most obvious sign of cataplexy (muscular weakness) involve the face and neck muscles.
3- Horses remain standing with their heads handing and resting close to or on the ground.
4- During an attack, flexion of the forelimbs may occur.
5- The eyes may be closed and occasional snoring may be heard.
6- When forced to walk, the horse may in coordinated as asleep complete attacks may occur and result in lying of the horse on its side with flaccid limbs. These episodes last from seconds to minutes.
7- The horse appears completely normal following these sleep attacks with no residual neuralgic deficits.
8- The stimulation require to induce an attack may vary from leading the horse out of the stall to strike the horse's back.

Diagnosis:

(I)History (II) Clinical signs

Treatment:

The administration of tricyclic antidepressant drugs as imipramine that block the uptake of serotonine and norepinephrine and suppresses only sleep. Imipramine is effective following IV or IM administration as 0.55mg/kg B.Wt relieving cataplexy for 5 hours and longer relief can be obtained with higher doses

Epilepsy

Definition:

Epilepsy means repeated seizures in which, the primary defect is in the brain

Etiology :

The primary defect may be:

1-Ischaemic 2-Traumatic

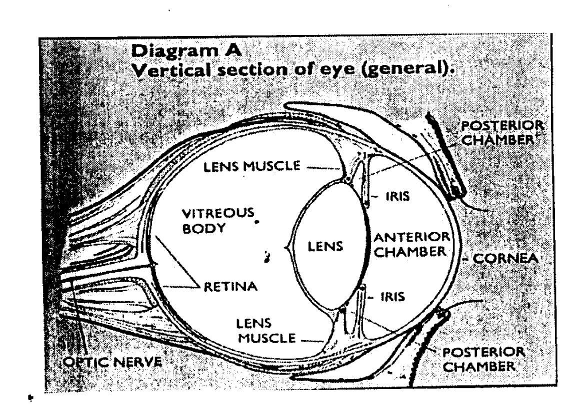
3-Infectious 4-neoplastic

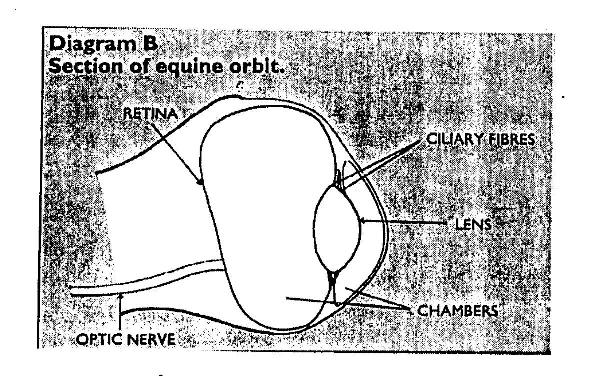
Lesions in epilepsy are mainly found in the cerebral cortex. Epileptic seizures appear after a time if the cause is traumatic as there is a delay from time of injury to the establishment of recurrent seizure due to the development of brain diseases.

Clinical findings:

1- The clinical characteristic of the epilepsy is the variability of signs with widely varying intervals between attacks, These intervals may be minutes. hours, days, weeks, months, or even years.

2- The seizure can include loss of consciousness, tonic and clonic muscular movement of the head and eyeballs jaw clamping. Opisthotnus, paddling actions and changes in visceral functions (urination, defecation. sweating and salivation)





Types of epilepsy

1-Idiopathic epilepsy

Epilepsy is described idiopathic when the cause is unknown

Etiology:

It has been observed in mares in association with injuries, high estrogen level; therefore reproductive and hormonal analysis should be considered in mares with seizure episodes. Progesterone therapy, ovariectomy or anticonvulsant therapy may prove beneficial in these animals.

Diagnosis:

1-Physical examination.

2-Neurological examination.

3-Cerebrospinal fluid analysis.

4-Skull radiography.

5-Blood chemistry.

Treatment:

Cases of epilepsy is treated with diphenylhydantoin

2- Epilepsy Following Brain Trauma

Head trauma is common in horses and may or may not accompanied by skull fractures. Contusions, lacerations, and hemorrhage can all a result of

traumatic insult. The most common site for intracranial hemorrhage is subarachnoid resulting in meningitis. Early-onset seizures after trauma have a better prognosis than those occurring after a delay. The brain lesion that may result from trauma includes meningiocerebral cortex or patchy areas of cortical atrophy or both.

3-Convulsive Foals

it is an idiopathic convulsive syndrome that occurs from several weeks to several: months of age. It is characterized by a sudden onset of recurrent seizures that may increase in frequency over a period of days to weeks so that the foal is convulsing many times a day. This case responds to anticonvulsive drugs.

Etiology:

The etiology is unknown.

Treatment:

Anticonvulsive drugs are recommended as phenobarbital 6mg/kg i.v. or diazepam 25-50 (0.05- 0.4mg/kg B.W) repeated after 30 minute intervals if necessary. Oral phenytoin has been recommended,

FOAL DISEASES



1- FAILURE OF PASSIVE TRANSFER (FPT)

Definition

FPT is considered when the IgG level of foal is less than 4 gm/L. Levels between 2-4 gm/L indicate partial failure. The disease is characterized clinically by high prevelance of septicemia and foalhood septicemia due to reduction of immunity.

Etiology

1- *Maternal factors:* problems with secretion of colostrum

a. Colostrum is produced with inadequate levels of immunoglobulins due to failure in the selective transfer mechanism in mammary gland to concentrate immune proteins from the blood before parturition.

b. Premature onset of lactation so that the Ig-rich secretion is lost before the foal sucks. This may occur in placantitis or placental separation.

c. Starting of lactation before parturition which occurs due to changing in hormonal levels.

d. Failure of milk let down in response to endogenous oxytocin or pain of the udder. The first case can be resolved by IV injection of oxytocin (1-2 IU)

e. Serious parturient conditions (e.g. rupture of cecum, or utero-ovarian artery).

f. Isosensitization of mare, which means carrying foals with neonatal isoeythrolysis. In this case the foal must be prevented from access of milk because milk contains harmful isoantibodies.

2- *Foal factors*

a. Premature foal: inability of intestine to absorb IGs

b. Intestinal malabsorption of IGs due to stress factors that hasten the time of closure of bowel to IGs.

c. Delayed ingestion of colostrum

i. Congenital anomalies: e.g. cleft palate.

ii. Delayed or impaired suck reflex

iii. Inability to stand due to weakness and limb abnormalities.

Diagnosis of FPT

1- Measure colostrum specific gravity: by colostrometer (hydrometer) : positive correlation exists between sp. Gr. and IGs content of colostrum

2- Protein precipitation: by using zinc sulphate (zn SO4) that gives opaque precipitation in the presence of IGs.

3- Specific immunoglobulin assay: using specific antiserum to detect the antigen-antibody conjugate as a precipitation.

Management of FPT

1- Foals under 12 hours old: are given colostrum (deep-frozen) 50 ml/kg by nasogatric tube while the small intestine is permeable to immunoglobulins.

2- Foals over 12 hours old: are given plasma (60 ml/kg) parenterally because the intestine is no longer open permeable to absorb IGs.

2- COMBINED IMMUNODEFICIENCY DISEASE

Definition

It is a genetic disorder of Arabian foals inherited as autosomal recessive trait. The disease is characterized by inability to produce lymphocytes and failure of immune response.

Clinical signs

Affected foal remains normal until maternal antibodies are catabolized to non-protective concentration. Foals then develop infections mainly of the respiratory tract, such as adenovirus and different bacteria

Diagnosis

1- Lymphopenia: less than 1000 lymphocyte per µl.
2- Absence of serum IgM.
3- Hypoplasia of lymphoid tissue as spleen and thymus

Treatment

No treatment is successful but euthanasia (humane killing) is indicated

3- NEONATAL MALADJUSTMENT SYNDROME



Fig. 1. A horse showing righting reflex

Definition:

It is a behavioral disturbance occurs at the first week of age characterized by convulsions, disorientation, incoordination and loss of sucking and righting reflexes.

Etiology

1- Early cutting of the cord may deprive the foal of large quantity of blood in the placental vascular bed.
2- Pulmonary dysfunction that results in deprivation of oxygen.

Clinical signs:

I- Two clinical stages are distinct:

a- Barking stage

1- Disorientation and coordination.
2- Loss of sucking reflex (pathognomonic).
3- Convulsions at face neck and shoulder.
4- Temperature is elevated during convulsion and reduced to room temp. at quiescence stage.
5- Galloping movement and barking sound.
6- Loss of righting reflex is the most obvious sign. The foal is unable to use the normal sequence of raising his head and flexing the limb in order to stand. The attempts to stand are followed by uncontrolled movements and the animal may bruise itself.

b- Dummy and wandering stage

1- Foals may be blind and does not respond to external stimuli.
2- Foals wanders or walk aimlessly.

II- Secondary signs: of GIT, respiratory and cardiovascular

Treatment

1- Anti-convulsive drugs: phenobarbitone sodium 20 mg/kg followed by a maintenance dose of 9 mg/kg BW every 8 hours.
2- Muscle relaxant and tranquilizer as Diazepam 0.6 mg/kg
3- Corticosteroids may be used as anticonvulsives such as dexamethazone 4 mg three time daily IV.
4- Assistant of the foal to raise his head and stand

4-LUNG ABSCESS IN FOALS

Definition

Development of single or multiple lung abscesses causing toxemia of foals between 2-4 months of age due to waning of maternal derived antibody levels and incomplete development of immune response caused infection with corynebacterium equi.

Etiology and pathogenesis

1- Corynebacterium equi infection may occur via aerosol as the MO is present in soils and spreads during dry season.
2- The MO adheres to cell wall preventing antibody production and inhibiting digestion within phagocytes.
3- The MO produces phospholipase and cholesterol oxidase enzymes that have a pathogenic significance.
4- The bacteria can live and multiplicate intracellular in neutrophils and phagocytes. Therefore, much of tissue destruction of pulmonary parenchyma is attributed to the lysosomal enzymes and free oxygen radicals released from degenerating neutrophils and macrophages.

Clinical signs

1- Dullness, anorexia and fluctuating fever.
2- Coughing is short and harsh.
3- Intermittent episodes of epistaxis and hemoptysis due to pulmonary hemorrhage following erosion of adjacent large pulmonary vessel.
4- When abscess is large, careful auscultation and percussion reveals circumscribed area of dullness over which no breath sounds are audible. Crackles (moist rales) can be heard at the periphery of lesion.
5- In multiple small abscesses, the dyspnea is more pronounced
6- Purulent nasal discharge and fetid breath occur only if bronchopneumonia developed from extension of the abscess.

Diagnosis

1- Case history
2- Clinical signs
3- Radiographic exam: reveals lung abcessation
4- Microbial culture of nasal or tracheal mucus may give false negative because the MO reside intracellular.
5- Blood analysis: leucocytosis.

Treatment

Treatment is mostly unsuccessful but a combination of eryhtromycine 25mg/kg three times daily and rifampin 5 mg/kg twice daily for 4-9 weeks may be effective.

5-NEONATAL ISOERYTHROLYSIS (NI)

Synonym: Isoimmune hemolytic anemia of the newborn

Definition

It is an immunologic disorder of newborn foals caused by reaction between serum antibodies of the mother and erythocytes of newborn foals due to incompatibility. The disease is characterized clinically by severe hemolysis and hemolytic anemia.

Incidence and Etiology

1- The foal inherits erythrocyte antigens from the sire (stallion) and these antigen pass through placenta into dam's circulation.
2- If the antigens are not also a part of the dam's normal complement, antibodies are produced in the dam' circulation against foal's erythrocytes.
3- The antibodies are produced by 8-10th month of pregnancy but can not affect fetus because they can not pass the placental barrier.
4- The antigen-antibody reaction occurs only when foal gets these antibody with colostrum.

Clinical signs

a. Severe haemoglobinuria and pale mucous membranes.

b. Jaundice.

c. Weakness and disinclination to suck.

d. Sternal recumbency.

e. Increase heart rate and increase amplitude of cardiac sounds.

f. Hyperpnea and dyspnea at later stage.

Diagnosis

1- Case history

2- Clinical signs

3- Lab. diagnosis:

a. Reduced RBCS count (<6 million).

b. Reduced PCV (<25%).

c. Reduced Hb level.

Therapy

1- Prevention of further ingestion of antibodies from colostrum by muzzling.

2- Corticosteroids may reduce the hemolysis: dexamthazone 5-20 mg IV.

3- Antibacterials: trimethoprim or ampicillin to reduce secondary bacterial infection.

4- Plasma transfer to restore blood volume

5- Whole blood transfer to replace the destroyed RBCs.

6- FOAL HEAT DIARRHEA

Definition

A self-limiting disease occurs in foals between 10-14 days of age that coincides with the mare's first postpartum heat or estrus.

Etiology

The exact cause is unknown. However, it has been found in the following conditions:

1- Hormonal changes within mares

2- Changes in milk composition
3- Over-engorgement with milk by foals
4- Bacterial or viral enteritis
5- Ingestion of genital discharge
6- Ingestion of roughage and irritant
7- Strongyloidosis (little proof)
8- Physiological changes within foal's GIT

Clinical signs:

1- Soft watery feces while foal is bright alert and nrses normally.
2- Temp. is within normal limit.
3- Dehydration may occur manifested by dry oral mucosa, sunken eye and shrinkage of skin.

Diagnosis

1- Case history
2- Clinical signs
3- Lab. diagnosis
   a. PCV and serum plasma proteins are elevated
   b. Fecal culture for antibiotic sensitivity.

Treatment

1- If foal is alert, bright and nursing normal , no therapy is required. Mineral oil or petroleum oil is applied topically to perineal region to avoid scalding.
2- If diarrhoea persists for long period:
   a. Anthelmintic administration such as cambendazole to control strongyloidosis
   b. Antibiotic based on culture and sensitivity.
   c. IV Fluid and electrolyte therapy to overcome dehydration.
   d. Mineral oil 180 gm and caster oil 30 gm by stomach tube to evacuate GIT.
   e. Administration of intestinal protectants.

SPORTS MEDICINE

THE EXHAUSTED HORSE SYNDROME

Definition:

Exhaustion is a result of brief maximal exercise or protracted (prolonged) submaximal exercise. With maximal exertion in racehorses, there is rapid depletion of readily available muscle energy stores (creatine phosphate, ATP). In this, exhaustion produces severe metabolic lactic acidosis. With protracted submaximal or endurance exercise, energy is supplied by aerobic metabolism of both fatty acids and carbohydrates with minimal changes in acid-base status or lactic acid concentration.

Etiology and pathogenesis

- The syndrome usually occurs during summer season, when the temp. and humidity are elevated.
- The syndrome is associated with 2 types of exercise high-intensity short exercise and prolonged submaximal exercise:

  a. **High-intensity short exercise:**

     i. This kind of exercise leads to rapid depletion of the phosphagen pool of muscle cells (creatine phosphate and ATP) and accumulation of lactate.

     ii. Accumulation of lactate reduces the muscle pH from normal (7.0 – 7.1) to 6:3 or lower.

     iii. The reduction of pH results in:

        1. Interfering with Ca availability by actomyosin, reducing the contractile efficiency.
        2. Inactivation of muscle metabolizing enzymes, thus reducing the availability and production of ATP.

  b. **Prolonged submaximal exercise:**

  Many factors- including disturbances in thermoregulation, hydration, and

ionic balance, and depletion of muscle glycogen stores- have been implicated:

i. As sweating is the most important mechanism for heat dissipation in the horse during prolonged submaximal exercise, large volumes of fluid may be lost via this route producing dehydration and reduction of exercise performance.

ii. Horse sweat is hypertonic, and sodium and chloride are the major ionic constituents of sweat. Sodium loss appears to have the most pronounced effects on body function. Due to the central role of sodium in fluid homeostasis, the major signs resulting from the loss of sodium and associated fluid are those of decreased circulatory function and poor organ perfusion.

iii. Loss of potassium ions in the sweat may occur and contribute to the exhaustion but usually transient because of the compensatory release from other cells.

iv. Calcium ions are required in the mitochondria of exercising muscles for excitation-contraction coupling process. This process requires oxygen, and therefore reduce the amount of oxygen used in the mitochondria for phosphorylation of ADP to ATP resulting in exhaustion.

v. Depletion of intramuscular glycogen store during prolong submaximal exercise contributes to lowering the exhaustion.

Clinical signs

1. There are variations in the severity of signs of exhaustion in individual horses.
2. All affected horses will have elevated rectal temperatures and pulse and respiratory rates, and variable dehydration on arrival.
3. The most reliable quantitative guides to impending exhaustion are pulse and

respiratory recovery rates. The pulse and respiratory rates take 30 minutes after rest and must return to acceptable levels, usually 60 to 70 per minute and 40 per minute, respectively.

4. Severely affected horses are usually severely depressed with little interest in food or water despite apparent dehydration.
5. Pulse pressure and jugular distensibility are often markedly decreased.
6. Capillary refill time is prolonged and cardiac irregularities.
7. Muscle cramps and spasms are often evident.
8. Diminution or absence of intestinal sounds.

Treatment

1- Horses manifesting depression and persistently elevated pulse and respiratory rates as their only problems may respond to rest, cooling out, and access to salt, clean feed, and water. A return of appetite and water consumption are exceedingly important factors in the recovery process. Horses should be closely watched and, if there is no improvement in 30 minutes, they should receive fluids orally or intravenously.

2- The rectal temp. should return to normal within 15-30 Min. to hasten this process, cold water is applied by a hose, sprayer, sponge, or towels over the large vessels of the distal extremities, head and neck, and over the jugular veins. This is most effectively done in an open area with free circulation of air, in a breeze or in front of a fan. It is a general practice to avoid pouring very cold water over the large muscle masses as there is concern of inducing muscle spasms. Cold alcohol leg wraps is also useful in dissipating heat.

3- The most important aspect of treatment of severe exhaustion is prompt and vigorous fluid therapy, the aims of fluid therapy are:

  a. Restoration of effective circulating blood volume: achieved by IV injection of saline (provides Na^+ and Cl^-).
  b. Correction electrolyte deficits by IV saline with addition of K^+.
  c. Provision of readily metabolizable energy as glucose.

N.B.

The use of non-steroidal anti-inflammatory drugs is controversial because they may produce toxicity in dehydrated, volume-depleted horses. However, they may be used after rigorous fluid therapy for their anti-inflammatory, analgesic and anti-pyretic effect.

POOR PERFORMANCE SYNDROME

Definition:

Reduced or inadequate performance among race horses and endurance horses with a history of previous good performance.

Etiology:

Common causes of poor performance are usually categorized by the body system they affect:

The musculoskeletal system (bones, joints, and muscle)

The respiratory system (nose, upper airways, trachea, lungs)

The cardiovascular system (heart, blood vessels, blood)

The nervous system (brain, spinal cord, nerves)

The gastrointestinal system (stomach, intestines

1- Musculoskeletal -

A- Degenerative Joint Disease

One of the most common causes of lameness in sport horses is degenerative joint disease (DJD), otherwise known as osteoarthritis. Areas that are often affected include the hocks (where it is known as *bone spavin*), the fetlocks, the pastern joints (where it is known as *ringbone*), coffin joints, and, less frequently, the carpal joints. Degenerative joint disease, as the name implies, is a disease of wear and tear. The joints are lined by specialized tissue, called hyaline cartilage that is vital to smooth joint function. This cartilage can become frayed and damaged due to the mechanical wear associated with exercise. The normally smooth glistening cartilage becomes eroded, exposing bone and causing pain.

Horses show lameness that may respond to rest initially, but usually worsens with time. Anyone with a knee problem can attest to the pain and recurrence of DJD

B- Navicular Disease

Disease associated with the navicular bone, which resides within the hoof capsule, has shortened the athletic career of many horses. The accepted causes included increased pressure within the navicular bone itself, and arthritis involving the navicular bone and surrounding structures such as tendons and the coffin joint surfaces.

C- Azoturia (Tying up Syndrome)

It causes a painful breakdown of muscle, which is often accompanied by a high heart rate and respiratory rate, sweating, and anxiety. The disease was traditionally called 'Monday Morning Disease', because it was seen in draft horses who were given Sunday as a day of rest but fed the full grain ration.

D- Equine polysaccharide storage myopathy (EPSM)

Has been shown to be a cause of tying up in some draft horses.

2- Respiratory

A- Inflammatory Airway Disease (IAD) (COPD)

In horses with IAD, there are more profound changes than just inflammation. Inflamed airways transform, becoming *hyperreactive*, or 'twitchy.' Essentially, they constrict more readily and to a greater degree than do the airways in normal horses. The symptoms of constriction (bronchoconstriction) are coughing and exercise intolerance.

The airways are thicker due to development of excessive tissue and inflammatory secretions, and thus have a narrower lumen (passageway). The narrower airways cause a bottleneck in airflow, especially during hard work, which in turn decreases the available oxygen that is needed for work.

B- Left Recurrent Laryngeal Neuropathy (LRLN, laryngeal paralysis, roaring):

For effective function, the larynx must be able to close while the animal is swallowing, and it must become very wide during maximal exercise, to afford the greatest airflow. Laryngeal paralysis, which is most common in horses taller than 16H, primarily affects the *left* side. For certain reasons, the *left recurrent laryngeal nerve* that serve the muscle that opens the larynx, begin to die. When nerve function is lost, the muscle is no longer stimulated, and so it atrophies. The end result is that the left side of the larynx becomes paralyzed and can no longer open normally. This prevents the horse from breathing freely during intense exercise.

C- Exercise Induced Pulmonary Hemorrhage (EIPH, or 'bleeding')

EIPH is very common in racing Thoroughbreds and Standardbreds .The pressure in the vessels of the lungs becomes so great in racing horses, that capillaries (very small blood vessels) in the lungs actually rupture.



Figure 1: Endoscopic view of blood in the trachea of a horse with EIPH

3- Cardiovascular Disturbances

- Atrial Fibrillation

With atrial fibrillation, the electrical signals that ordinarily pass from the atria, the first set of filling and pumping chambers of the heart, to the ventricles, the second set of chambers, becomes disorganized. This causes the atria to beat in a very erratic fashion.

Because the ventricles still beat normally despite the erratic signals sent to them, the heart is able to pump adequate amount of blood at rest. But the extra little blood pumped in by the atria, while insignificant during rest, becomes important for the horse to perform strenuous exercise. For this reason, atrial fibrillation may go undetected for a very long time in horses that do light work, such as pleasure horses, trail horses, and show hunters.

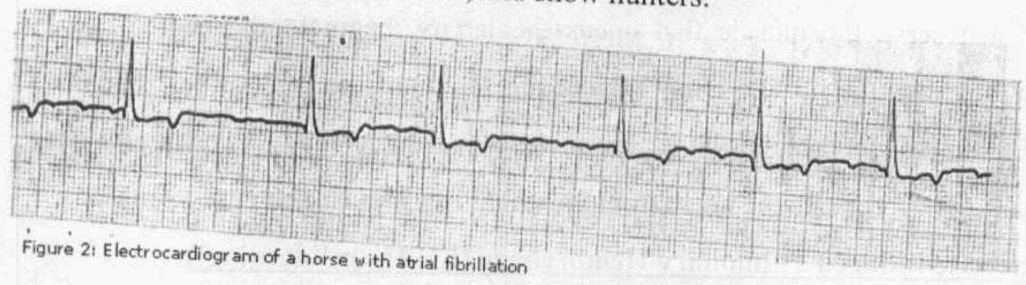


Figure 2: Electrocardiogram of a horse with atrial fibrillation

4-Neurologic Disturbances

A- Equine Protozoal Myeloencephalitis (EPM)

EPM is caused by a protozoal parasite (*Sarcocystis neurona)* that invades the neural tissues of the horse. The spinal cord is most frequently affected, but the brain may also be involved The most commonly seen abnormalities include ataxia (lack of coordination), muscle atrophy (especially of specific muscles in the head and tongue, as well as the gluteal and quadriceps muscles).

B-Cervical Vertebral Myelopathy (CVN)

CVM causes neurologic symptoms due to problems in the neck. The bones in the neck have a deformity or instability that pinches the cord.

5- Gastrointestinal disorders

- Gastric Ulceration

Horses with gastric ulceration may show varying signs, such as poor appetite, chronic colic, poor performance, 'crabby attitude', and teeth grinding.

Treatment

Treatment according to the etiology:

1. Inflammatory Airway Disease (IAD) is best treated with a combination of environmental management, anti-inflammatories (corticosteroids), and judicious use of bronchodilator drugs.
2. Left Recurrent Laryngeal Neuropathy is treated with surgery.
3. Exercise Induced Pulmonary Hemorrhage is commonly treated with Lasix, which is a diuretic. Studies have shown that Lasix may improve performance due to its effect to cause the horse to urinate, and thus shed 15-30 lbs of body weight right off the start. Atrial fibrillation is treated with a drug called *quinidine*. Most horses with benign atrial fibrillation will respond favorably to quinidine administration.
4. Equine Protozoal Myeloencephalopathy must be treated with anti-protozoal drugs. The most commonly used combination is *pyrimethamine (Daraprim)* and sulphonamides.
5. Cervical Vertebral Malformation - there is no real cure for CVM. Surgery to stabilize the vertebral column has been attempted, but full return to athletic function should not necessarily be expected in any case.
6. Gastric ulceration should be treated with a combination of management changes (more roughage, less concentrate in the diet), and drugs that decrease acid production in the stomach.
7. Degenerative Joint Disease can be treated with a combination of training changes (usually, decreasing the concussive workload), anti-inflammatories such as phenylbutazone and corticosteroids (corticosteroids should be reserved for low motion joints such as the lower hock joints), and physical therapy (warm water soaks before work, cold water after, massage, range of motion exercises).

8. Navicular disease is initially treated with shoeing changes. Phenylbutazone and drugs that are thought to increase blood supply to the area may also be recommended. In chronic cases that do not respond to conservative treatment, a *neurectomy* (cutting the nerves that supply sensation to the heel) may be recommended. Vasodilators , also seem to reduce pain in some horses.
9. Exertional Rhabdomyolysis. The horse may require intravenous fluids, anti-inflammatories, and sedation.

SYNCHRONOUS DIAPHRAGMATIC FLUTTER (SDF) (THUMPS)

Definition

It is a contraction of diaphragm that is synchronous with heart beats, which may be left-sided, right-sided or bilateral.

Etiology

1- Electrolyte imbalance due to digestive disturbances.
2- In horses affected with lactation tetany (transit tetany).
3- Following administration of furosemide as a premedication for exercise induced pulmonary hemorrhage.
4- Oral administration of excessive amount of sodium bicarbonate to volume –depleted and chloride depleted horses.
5- Anatomical alterations of phrenic nerve or its myelin sheet.

Pathogenesis:

1- SDF is usually associated with atrial depolarization.
2- The acid base and electrolyte imbalance may alter the membrane potential of phrenic nerve allowing it to discharge in response to the electrical impulse generated from myocardial depolarization.
3- The metabolic alkalosis results in binding of ionized calcium to plasma proteins. The decrease in ionized calcium leads to an increase in neural

irritability. This change of neural irritability in the phrenic nerve leads to it being stimulated by the polarisation-depolarisation cycle of the cardiac muscle. This results in the diaphragm contracting in response to the heart contractions; hence a diaphragmatic flutter; synchronous with the heart beat. This is commonly referred to as thumps since the flank muscles often respond with a hiccup-like "thump".

Clinical Signs

1- Twitch or contraction in the flank region, which may be unilateral or bilateral.
2- The contraction may be severe producing thumping sounds
3- The contraction is synchronous with heart beats.
4- Horses with SDF are usually dehydrated and volume-depleted.

Treatment

1- Injection of 100-300 ml of 20 % calcium borogluconate slowly IV with monitoring heartbeat produces prompt response.
2- Correction of the metabolic alkalosis and hypokalemia

PARASITES OF HORSE

General information :

Internal parasites are the greatest single cause of horse colic beside their role as a causative or contributing factor in many of respiratory digestive and performance problems.

Although parasitism of horse is the constant problem for horse owners great disease in their severity could be obtained by regular deworming program (preventive worming program)

There are 4 common internal parasites in horses and donkeys. These are: Gastrophilus equi (Bot) Strongylus, Ascarids, and Pin worm.

1- Bots

(Gastrophilus equi larvae)

Bots are larvae (immature worm) of botfly. They infect almost all horses. Mules and donkeys because the fly is common in horse environment.

Life cycle:

The flies (during hot or warm months) lay their eggs on the hair of various parts of the horses body such as nose, Chest throat and far legs. This stimulates the horse to lick the body and so carry the eggs on the tongue

The eggs hatch to larvae which enter the mouth and tongue. After one month, the larvae migrate to the stomach. In the stomach, hundreds of the larvae attach to the stomach lining causing irritation, interference with digestion and obstruction to the pyloric orifice to the small intestine after 8-10 months. The larvae passes with faeces to outside. They burrow into the mud or ground to pupate. During about 1 month. The adult fly repeats the cycle again.

The deworming program should continue all the time in our country as bot flies are killed by freezing temperature. In cold countries the deworming program could be stopped after the hard frost.

Several commercial: anti-bat prepâration are on the market such as

Commercial name	Active principle	Dose and route of injection
1-equivalan (.paste)	Ivermectin	0.2mg/kg B.W.S/C
Trichlorofen	Trichlorofen	40mg/kg B.W orally
3-Carbon	Carbon disulfide	2.5ml1/5 Kg B.W orally

2-Ascarids

(Large Round worm)

These round worms are 15-23 cm length are usually found in hand reds in the small intestine of horses. Mules and donkeys interfering, with their nutrition and may cause colic cough and diarrhea these worms Usually after young horses more than adult ones.

Life cycle:

Infective ascarids eggs (infective stage) are swallowed with contaminated hay or water. They hatch in the intestinal tract. The larvae burrow through the intestinal wall where they reach the lung within one week from the lungs it migrates up to the trachea to the mouth where it reswallowed again to intestine. They become mature in the intestine in 2-3 months, then they lay up to200.000 eggs/ female that passed with the faeces to contaminate hay and become infective egg.

Foals should be first treated at 8 weeks of age and retreated every 6-8 weeks until 2 years ago.

The effective treatment or preventive program may be achieved by using the following preparations.

Commercial name	Active principle	Dose and route of injection
1- Thiabenzole	Thiabendazole	80-100mg/Kg B.W. orally
2- Equivalan (paset)	Ivermectin	0.2mg/Kg B.W. orally with sugar
4-tyvert	Oxyfenbendazole	1 cm/5 Kg B.W. orally
5- Fenbendazole	Fenbendazole	5mg/Kg B.W. orally
6-Vermavet	Mebendazol	1 gm/10Kg B.W. orally
7- Piperazine citrate	Piperazine citrate	3g/10Kg B.W. orally

3-Strongylus

This term refer to large group of closely related species of internal parasites they also called blood sucking parasites or blood worms these worms are very dangerous because the larvae (immature worms) migrate via blood vessels of intestine causing inflammation of intestine which may result in sever fatal colic Horses mules and donkeys of all ages are infested life cycle.

The female worm in the intestinal tract eggs which passes with faeces. Favorable environmental condition (warm and moisture conditions) the eggs hatch into larvae in the manure. The egg may resist dryness and coldness of long period without hatching. The infective larvae migrate to green grass to bee ingested by grazing horse. In the intestine the larvae develop into young parasite and migrate for 6-7 months along the larvae develop into young parasite and migrate for 6-7 months along the walls of arteries. Liver and intestinal wall. They reurn to the large intestine as young adults. The adults lay up to 5000 egg/ female/ day in the large intestine that can pass the faeces to complete the life cycle.

Symptoms :

The main are loss of condition weakness and diarrhea Anemia is a main symptom due to blood sucking parasites. Horses in apparently god physical condition may have large numbers of strongylus larvae that can create arterial aneurysms which easily rupture the blood vessel resulting in sudden death due to internal bleeding.

So Routine faecal examination beside the routine deworming program are essential and effective tools for prevention. Blood examination needed to determine the severity of the case i.e. Hb%, PCV, RBCs count. The effective preventive program may include the use of:

Commercial name	Active principle	Dose and route of injection
1- Thiabenzole	Thiabendazole	80-100mg/Kg orally
2- Equivalan (past)	Ivermectin	0.2mg/Kg B.W. S/C
4-tyvert	Oxyfenbendazole	1 gm/Kg B.W. orally with sugar.
5- Fenbendazole	Fenbendazole	1 cm/.5/Kg B.W. orally
6-Vermavet	Mebendazol	5gm/10Kg B.W. orally
7- Piperazine citrate	Piperazine citrate	3gm/10Kg B.W. orally

3- Pinworms

(Oxyuris equi)

Commercial name	Active principle	Dose and route of injection
1- Thiabenzole	Thiabendazole	80-100mg/Kg B.W. orally
2- Equibvalan (past)	Ivermectin	3mg/10Kg B.W. orally

Equine Euthanasia
(humane killing)

It is difficult and serious decision to take for a horse. The horse usually had a very special and emotional relation to the owner himself, the owner's family and the horse attendant.

It is not enough for the horse to be so sick, severally injured old or lame to give our decision to inform the owner that his horse should be quickly and humanely euthanasized. The reaction will be varied from pain in experienced owners to refusal in non-experienced one. So you should explain the emotional factors in details at first then the financial cost that will be needed for the horse to be alive